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Editor: H. A. Shapiro, Ph.D., M.B., Ch.B., F.R.S.S.Af.

The Brussels Congress on Forensic Pathology

The First International Meeting on Forensic Pathology, held in Belgium from 19-21 July, was extremely well attended and thoroughly representative of the global interest in this field. Members were present from Algiers, Belgium, Denmark, Eire, France, Great Britain, Italy, Poland, South Africa, the Sudan, Sweden, the United States of America and Yugoslavia. There was also a registration from Germany.

Dr. Charles Larson from Tacoma, Washington, President-Elect of the American College of Pathologists, was the President of the Brussels Meeting and the following were Vice-Presidents:

Dr. P. Enderlé (Belgium), Dr. S. Faulds (Great Britain), Prof. John Glaister (Great Britain), Prof. P. Moureau (Belgium) and Dr. H. A. Shapiro (South Africa).

Dr. Francis Camps of the London Hospital Medical College and Prof. F. Thomas of the University of Ghent, acted as Secretaries. The Meeting is deeply indebted to them for the efficient way in which they arranged the programme. All those who attended carried away the warmest recollections of the most generous hospitality which the Belgian hosts showered upon the visitors.

A variety of interesting and original papers was discussed very fully, the discussion on each paper being summarized in the alternative language of the Congress (English or French). The brochure on the Congress contained bilingual summaries of all the papers submitted to the Congress and thus facilitated the conduct of the International Meeting very considerably. The proceedings of the Meeting will probably be issued as a volume embracing the discussions.

A business meeting held during one of the lunch hours made quite clear the unanimity of opinion that further international meetings should be held at intervals of two or three years. No formal constitution or procedure was adopted, as there was no desire to create yet another organization or institution. The arrangements for the next meeting, which will be announced in good time, can confidently be left to the sponsors and the capable executive officers who arranged the Brussels Meetings with such success and distinction.

The Medical Aspects of a Case of Manual Strangulation

G. S. W. de Saram, *O.B.E.*, M.B., B.S.
Department of Forensic Medicine, University of Ceylon, Ceylon

At about 3.15 p.m. on 9 October 1951, the dead body of Mrs. X, with a mortar over its neck, was found in the garage of the house in which she lived.

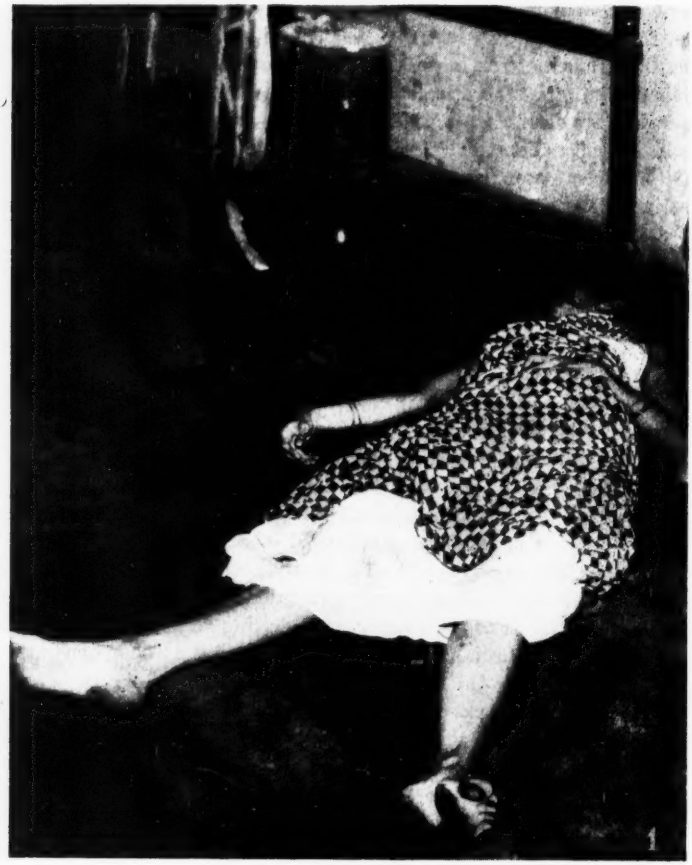

Fig. 1. The body in the garage at the time of the medical examination. (Note inverted mortar and the billet of firewood to the right of the body.)

The body lay on its back on the cement floor of the garage with its legs extended and its feet 16 inches apart (Fig. 1). It was clothed in a cotton petticoat and an old artificial silk saree 6 yards long, several folds of which were, as usual, draped round the body in the form of a skirt and secured by a pin at the waist. A short length of the saree, passing over the left shoulder and the back, was secured to the right waist. The arms were bare and the saree and the cotton petticoat were drawn up just below the knees. Dirt was adherent to the soles of both feet and especially to the pads of the toes of the right foot and the front of the sole (ball) of the left foot to which a flake of dirt was attached. The heavy mortar, described as being originally on the neck of the deceased, and a billet of firewood were, at the time of the medical examination, on the floor to the right and somewhat to the rear of the body. A pearl similar to those inset in the deceased's earstuds, one of which was missing, was on the floor not far from the body. A hand-mirror, later established as belonging to the deceased, was on a stool in the garage.

The garage (which had a concrete roof) was at one end of the house. It opened on to a lane in front through a door which was usually kept closed. The door was trellised and curtained with jute-hessian in its upper part. From the rear of the garage a few winding narrow steps, flanked by walls on either side, gave access through a doorway 18 inches wide to what was later in the course of the trial referred to as the 'Dover-stove kitchen'. This in turn communicated with the 'gas-stove kitchen'.

A pot of rice, still warm, was found on the cooker in the gas-stove kitchen, while there was other evidence of preparation for lunch in the Dover-stove kitchen. On the floor of the latter there was a coco-nut grater[1] with half a husked coco-nut nearby; and what was described by the Government Analyst as a drag-mark, about $1\frac{1}{2}$ feet long and $1\frac{1}{2}$-$2\frac{1}{2}$ inches broad, wavy in outline and about $1\frac{1}{2}$ feet from the door-way leading to the garage.

The gas-stove kitchen opened on to an open back verandah which led into the dining-*cum*-sitting room of the house. Through the front hall one could proceed up a flight of stairs to the bedrooms.

The bedroom occupied by the deceased and her husband was in disarray. The remnants of the morning meal consisting of 'string hoppers',[2] etc. were on a stand in the room. When first seen by the ayah[3] that afternoon the wardrobe in the bedroom was open, with the lady's handbag inside the wardrobe and the keys on the floor. A fragment of firewood was found on the stairs.

At the time of the discovery of the body the 2 younger children of Mrs. X (both under $4\frac{1}{2}$ years) were alone in the house. It was they themselves who had directed a neighbour, calling to use the telephone, to the garage. They were joined almost immediately by Mrs. X's 2 elder children

and the ayah, who had been for over 40 years in the deceased's family, on their return from school.

The police investigation began almost immediately, the police station being quite close to the house.

When the ayah and the 2 older children left at 8.15 that morning, there were in the house, besides the 2 younger children and the deceased (who was then reading the morning papers), the husband of the deceased (who was asleep in the bedroom) and a young man-servant of about 18 years who was in the kitchen. At the time of the discovery of the body, however, both these men were missing. According to the ayah the man-servant had been her mistress' employee for about 10 days, and was well-behaved. Mrs. X had filed a divorce action against her husband, who had returned from abroad about 18 days before the tragedy. The evidence of Mrs. X's relatives was to the effect that she and her husband had been partially reconciled, but that the action had not been discontinued.

The husband was found at about 8 p.m. that evening at the house of a friend in Colombo. On examination that same evening no injuries were found on any part of his body. His finger nails were clean and about 1/16th of an inch long. The young servant was located, 9 days later, about 150 miles to the south of Colombo. He had 4 healing finger-nail marks on his left cheek and 2 similar nail marks on his left forearm.

In the Trial Court about 18 months later the husband was charged with having murdered the deceased. The servant, a co-accused in the lower court, was granted a conditional pardon by the Crown at the trial, his evidence forming the basis of the case for the prosecution.

There were 2 points of time which could be fixed by the evidence of reliable witnesses, namely 8.15 a.m. on the one hand and 10.30 a.m. on the other.

The ayah spoke to the events leading up to 8.15 a.m. and described the state of the household when she left with the 2 older children for school that morning. The evidence of the ayah on the relevant points was briefly as follows:

That morning the 2 older children, who were going to school later, had their morning milk at 7 a.m., Mrs. X as usual had a cup of coffee and milk. At 8.15 a.m. a taxi, summoned by telephone, arrived and took the ayah and the 2 older children to school. At 8.15 a.m. Mrs. X had not had her morning meal and when the witness and the children left, Mrs. X was seated in the children's room reading the newspapers. The younger children had not yet been fed and the string hoppers were in a parcel wrapped with newspaper in the children's room. The husband was asleep in bed, the witness having seen him when she went into the bedroom to ask the deceased to telephone for a taxi.

What occurred after 10.30 a.m. is also quite clear. At 10.30 a.m. a

taxi, also summoned by telephone, arrived at the house and the husband left and was later found in the company of persons who gave evidence at the trial.

The case for the Crown was that between 9 and 9.30 a.m. Mrs. X was murdered by her husband, who had sought assistance of the servant.

The evidence of the servant was broadly to this effect:

The witness was grating a coco-nut in the kitchen that morning when the accused came up to him and asked him to assist the accused in killing the lady of the house. On their arrival at the bedroom the accused,

Fig. 2. A coco-nut scraper in use.

1. A low stool on feet, 4-6 inches high, with a serrated circular metal disc fixed to one end (Fig. 2).
2. A steamed preparation of rice flour, similar to riced potato, in the form of cakes.
3. Nurse-maid.
4. According to Hindu custom, the substitute for the wedding ring. Placed round the neck of a Hindu bride by her husband at the nuptial ceremony, it consists of a metal container (usually of gold) holding sacred symbols blessed by the priest, attached to a gold chain.

approaching the deceased (who was seated on the bed) from her right-hand side, grasped her by the hair and throat with his left and right hands respectively and dragged her down on to the floor. He shifted his position then to the left-hand side of the lady and, while kneeling and using both his hands for the strangling, called on the witness for help. The witness then steadied the victim's hips with his hands, and it was while he was doing so that the deceased had inflicted the injuries found on him. The accused next stamped with his rubber-soled shoe on the neck of the victim, and the witness then knew that her voice-box had broken as her head had turned to one side. The accused thereupon took some money from the deceased's hand-bag and gave it to him together with her thalikody,[4] 2 gold bangles, and a ring removed from the body of the deceased. The accused closed the front door and the 2 of them carried the deceased downstairs to the garage. The witness was definite that while doing so he himself held the deceased's legs, and that at no time did the feet of the deceased come in contact with the floor. He had left the house about 9.30 a.m.

The prosecution then alleged that, after the servant's departure, X remained in the house with the corpse and his 2 young children; that he daubed the feet of the corpse with dirt off the kitchen floor to further fix the murder on his accomplice; that he went upstairs for the hand-mirror which he used to satisfy himself that life was extinct; and that he introduced an unknown third person who had summoned the taxi by telephone, and waved to him from the front door when he left the house at 10.30 a.m., fully one hour after the departure of the servant.

The position of the defence in answer to the prosecution case was that when the accused had left the house at 10.30 that morning the deceased was still alive and had in fact waved goodbye at the front door (as was testified to by the taxi-driver who recognized her); that death had not occurred *before* 9,30 a.m. and could only have occurred *after* 10.30 a.m. that the strangling had taken place while both victim and strangler were standing in the kitchen; and that it had been effected by the servant only, as proved by:

1. His account of 'the breaking of the voice-box' which could only have come to his knowledge had he himself been the strangler.
2. By the finger nail marks on his face and arms, which had admittedly been caused by the deceased.

On the question of the time of death the Prosecution and the Defence were at variance, the purpose of the Prosecution being to prove that death had occurred before 9.30 that morning, while the Defence set out to establish that death had occurred after 10.30 a.m. On the question of the manner of strangling, too, and as to events subsequent to the strangling, the two cases indicated a wide divergence of view. In the absence of

evidence (other than the evidence of the servant) the emphasis of the case rested upon the medical testimony.

The Prosecution relied from the lower court onwards upon the opinions of 2 surgeons, Professors of the University of Ceylon, of a Government Radiologist and of the late Dr. John Taylor, of St. John's Hospital, London (who was in Ceylon as an examiner in the primary F.R.C.S. examination which was being held here at the time). Dr. Taylor had unfortunately died by the time the case came up for trial. Professor Sir Sydney Smith was cited as a witness for the Defence and gave evidence in the trial court.

At the trial the medical issues raised were as follows:

(*a*) When did death occur?

(*b*) How did it occur?

(*c*) Where did it occur?

(*d*) What were the events subsequent to the strangling?

The Time of Death

The medical deductions about the time of death were based on:

1. The cooling of the body, calculated from the rectal temperature.
2. The degree of rigor mortis.
3. The contents of the stomach and small intestine.

Estimation of the Time of Death from Cooling. The rectal temperature when taken at the scene at 6.55 p.m. was 93.2°F. and the temperature of the room 81.5°F. In the estimation of the time of death it is usual to adopt a formula such as:

$$\frac{98.4^\circ\text{F. (Taken as Normal Temperature)} - \text{Temperature Observed at Time of Initial Examination}}{\text{Hourly Rate of Fall}} = \text{Hours after Death.}$$

We have found that in the corpses of normal adults in the low country, as around Colombo, the hourly rate of fall usually lies between 0.7-0.8°F. in observations made about 5-8 hours after death. As, however, cooling may be slower in asphyxial death the rate used was 0.7°-0.6°F. per hour. The probable time of death by this method was therefore estimated as being 7½-8¼ hours before examination, i.e. between 10.15 and 11.30 a.m.

Estimation of the Time of Death from the Gastro-Intestinal Contents. We have observed that the stomach starts emptying within 10 minutes of the ingestion of the first mouthful of a solid meal and that the head of the meal progresses along the small intestine on the average of 6-7 feet per hour, reaching the end of the small intestine in about 3-3½ hours. The contents of the stomach, viz. about 10 oz. of fluid material, sedimenting down on standing for one hour to about 3 oz. of solid material, consisting

chiefly of string hoppers, was considered unreliable for any deductions to be made because:

1. The quantity of the food originally consumed was unknown.
2. The possibility that the meal had been staggered.

It will be remembered that the finding of the meal did not suggest a meal consumed at one sitting. The fluid found in the stomach, besides, might have been independently ingested at any time before death. The meal in the small intestine was continuous with that in the stomach and consisted of partially digested string hoppers. The head of the meal had reached to about 3 inches from the end of the small intestine. The time of death, as estimated from the progress of the head of the meal was, therefore, fixed as being probably 2-3 hours after the commencement of the meal. According to the ayah, the meal was certainly not eaten by the deceased before 8.15 a.m., the time she had left with the children. Death had probably occurred, therefore, between 10.15 and 11.15 a.m., there being no pathological changes in the intestine or the stomach to suggest changes in the rate of emptying.

The Estimation of the Time of Death by Rigor Mortis. The presence of established rigor mortis in the whole body and, to a slight extent in the fingers and toes, indicated the time of death as lying possibly between 8-10 hours previous to 5.55 p.m., when the condition was investigated. Rigor is uncertain in its onset and progress. But we have found its manifestation in the pupillary muscles of the eye and the degree of transparency of the cornea useful in the interpretation of the time of death as assessed from rigor of the body as a whole. The semi-dilated condition of the pupils in the presence of a clear cornea in this case would correspond to 5-7 hours after death. The time of death as estimated from rigor was therefore considered to be about 10 a.m.

The limits of probability of the time of death assessed from all factors were given, shortly after the post-mortem examination, as lying between 10 and 11.30 a.m. In the lower court, in answer to a question by the Prosecution, it was stated that an extension of the time to 9.30 a.m. was just possible and a similar extension to later than 11.30 a.m. was less likely.

It would appear that this estimation of the time of death was challenged by the Prosecution on the following grounds:

Cooling Rate: The estimation of the time of death from the cooling of the body, it was suggested, was incorrect because, although the hourly rate of fall of 0.7°-0.6°F. was accepted, the initial rectal temperature was considered to have been higher than 98.4°F., for 2 reasons:

1. The rectal temperature is usually higher than 98.4°F.
2. Violent physical or convulsive exertion would physiologically have tended to raise the body temperature.

Our estimate of 0.7°-0.8°F. was based on an initial rectal temperature

of 98.4°F. To presuppose a higher initial rectal temperature, effecting the numerator, with no commensurate change in the hourly cooling rate, in the denominator of our formula, would be fallacious, for it is accepted that a dead body cools on the principle that the greater the body—room temperature difference the faster the fall. It was, besides, particularly unlikely for a slightly-built woman (weighing approximately 90 lb.) to have put up any degree of resistance against such a stranglehold involving rapid unconsciousness from pressure on the carotid sinuses and arteries. In any event, by the adoption of a 0.6°F. rate of fall per hour such contingencies, it was considered, would have been covered.

Gastro-Intestinal Contents: The Government Radiologist who was called in by the Prosecution stated that at the instance of the Police he carried out certain experiments to determine radiographically how the stomach empties when a particular type of meal is consumed. For the purpose of this experiment he was provided with a meal of particular composition consisting of a specific number of string hoppers, etc. decided on by the Police. Mixed with barium and malted milk, he gave this meal to 4 women (who were also provided by the Police) and watched the progress of the meal along the stomach and intestines. He described his experiments in great detail but did not prove to be of any help to the prosecution case because he agreed, under cross-examination, that the head of a meal such as the one given to the subjects of his experiments would take 2—3 hours to reach within 3 inches of the small intestine.

Rigor Mortis: Both surgeons agreed that rigor was an unreliable sign but that the assessment of death from rigor as lying between 8—10 hours after death was reasonable. The constriction of the pupils, they contended, was an unreliable test.

Estimation of Time of Death from All the Factors: Of the medical opinion called by the Prosecution, Dr. Taylor (who gave evidence in the lower court) was of the opinion that death would have taken place between 5.30 a.m. and 10 a.m. One of the surgeons placed it between 7.25 a.m. and 10.19 a.m. and the other between 7.42 a.m. and 11.30 a.m. The deceased, however, was reading the morning papers, not having had her morning meal, at 8.15 a.m. Therefore, as was stated by the trial judge, relating these views to the facts, the opinion of these medical men was that death *may* have taken place any time between 8.15 a.m. and 11.30 a.m. None of them, however, stated that it *must* have taken place before 9.30 a.m.

At the suggestion of Sir Sydney Smith certain experiments were conducted (in the University) shortly before the trial with a view to observing the post-mortem cooling rate in similar circumstances, and factual statements on these observations were communicated to him.

Briefly the nature of the experiments and the results thereof were as follows:

The dead bodies of 3 prisoners who were judicially executed at 8 a.m. on different days were clothed in a petticoat and a saree of material similar to that worn by the deceased. Half-hourly rectal temperature observations were recorded while the bodies lay on the cement floor of a room. Table I furnishes data about physical build, etc. as these are modifying factors of the cooling rate.

The last column indicates the number of hours which elapsed before the rectal temperature reached 93.2°F.

Table 1.

Body	*Weight*	*Height*	*Range of Room Temperature*	*No. of Hours since Death*
No. 1	112 lb.	5′ 1″	81·0° F.—83·5° F.	6½
No. 2	152 lb.	5′ 5″	79·0° F.—82·0° F.	7½
No. 3	130 lb.	5′ 3″	80·0° F.—83·0° F.	8½

As compared with the above, the corresponding details in the case and the estimated time of death are:

Mrs. X	90 lb.	5′ 3¼″	81·5° F.	7½—8¾

The estimation of the time of Mrs. X's death from all factors had been given as being between 7½—9 hours before 6.55 p.m., i.e. between 10 a.m. and 11.30 a.m.

The cooling rate is generally accepted as being the most reliable criterion in the estimation of the time of death. In the light of these tests, the limit of possibility was fixed at 10.30 a.m. and the compass of probability between 11 and 11.30 a.m.

The Manner of the Attack

The Prosecution said that the deceased had been strangled on the floor in the bedroom. The Defence maintained that it had been effected while the deceased was standing in the kitchen.

That death resulted from strangulation was not disputed. Our reconstruction envisaged strangulation being effected while assailant and victim were standing. Operating from behind, the killer's left hand had gripped the victim's left lower jaw and the right side of the voice box. The right arm passing behind and around the victim's neck enabled the fingers of the right hand to grip the left side of the voice box, while the right thumb reinforced the grip of the fingers of the left hand on the right side of the voice box. The victim had been effectively immobilized by pressure on the left collar bone and shoulder from the killer's left forearm, her left foot being pinioned to the floor by pressure from his left foot placed over her own left foot. An attempt had been made by the victim to tear away the

hold by insertion of the fingers of the right hand over the upper border of the killer's right hand, which would have been occupying a position low down the neck.

The relevant injuries, all ante-mortem, were:

1. An abrasion $1\frac{3}{4}$" × $\frac{1}{2}$" over the left lower jaw extending to the left from the middle of the chin along its lower border.

2. Half an inch above the middle of injury No. 1 was a linear superficial abrasion $\frac{1}{4}$" long and lying obliquely.

3. Half an inch behind and $\frac{1}{4}$" above the rear end of abrasion No. 1 was an abrasion 2" long and $\frac{1}{2}$" wide extending horizontally forwards from the left angle of the lower jaw. The soft skin over this abrasion was depressed and fragments of fine dark powder were found along it and on the jaw below it.

4. Lying obliquely over the right side of the voice box was an abrasion $1\frac{1}{4}$" long × $\frac{1}{8}$" wide consisting of some irregular scratch marks (Fig. 3).

Fig. 3. The irregular scratch marks described under injury No. 4.

5. One inch to the left of the lower end of injury No. 4 was an abrasion ½″ × ⅛″ wide consisting of 3 linear scratch marks ⅛″ long and a localized spot 1/16″ in diameter.

6. One inch below the middle of the left lower jaw was an abrasion ¼″ in diameter.

7. A superficial abrasion ⅛″ × 1/16″ on the left mastoid region immediately behind the ear-stud of the left ear.

8. On the left side of the neck 1″ from the middle front were 3 abrasions 1/16″, 1/16″ and ¼″ in diameter at intervals of ¾″ and 1½″ and in line.

9. Several parallel linear abrasions 2″ to 1″ long over the lower front of the neck.

10. An abrasion 1″ × ½″ over the middle of the left collar bone.

11. An abrasion 1″ × ½″ over the front of the left shoulder.

Corresponding to wound No. 9 there was a contusion 3″ × 2″ × 1″ deep over the lower front of the neck. There was much contusion around the voice box in relation to the superficial injuries on the neck and both superior horns of the thyroid cartilage of the voice box had been broken.

Injuries Nos. 6 and 8 on the left of the voice box indicate the characteristic finger-nail marks (four) of a right-hand grip. The absence of the usual abrasion associated with the thumb nail, high up on the neck to the right of the voice box, may probably have been due to the fingers of the killer's left hand intervening between thumb and neck. The scratch marks in injury No. 9 may possibly have resulted from some part of the thalikody intervening between his right hand and the victim's neck. The contusion underlying this injury led to the inference that the pressure exerted by the lower border of the right hand had been considerable.

The left-hand grip was deduced as follows:

Wound No. 1 was traced to the left index finger placed under the chin, the thumb pad of the left palm corresponding to wound No. 3. Wound No. 2, which was quite separate, would correspond to the outer border of the nail of the left thumb, which would naturally be flexed in the process. The position of the middle, ring and little fingers of the left hand, it was deduced, would correspond to the upper 3 marks, with their concavities facing to the left, described under wound No. 4. The lower 3 nail marks (2 of them with concavities facing to the right) were inferred to have been caused by the nails of the victim's right thumb, index and middle fingers as she attempted to tear the killer's right hand away with her own right hand. Injuries Nos. 10 and 11 were attributed to the pressure applied by the killer's left forearm and elbow as it lay over these parts. Injury No. 7 probably resulted from pressure by the killer's left hand on the ear-stud, which was found bent.

The dark powder (some of which had to be detached with the finger nail), adherent to the cheek and lower jaw in relation to wound No. 3, was subsequently examined by the analyst and found to resemble coco-nut fluff. It may have been left there by the hand of the assailant when it was

applied to this part of the face, had the hand previously been contaminated with fluff. It was suggested that the adherent coco-nut fluff together with injury No. 9 and the underlying contusion may have resulted from the stamping of the face and neck, as alleged by the servant. The shoes of the accused, however, had not been examined. The further possibility that the fluff from the coco-nut husks found in the garage in close relation to the head being deposited on the jaw by air currents was discounted as the fluff adhering to the face was firmly embedded.

The prominence of the dirt on the pads of the toes of the right foot and the forepart of the sole of the left foot, led to the inference that the left foot of the deceased had probably been firmly compressed against the kitchen floor while she attempted to further supplement her efforts to escape by rising on the toes of the right foot. This was further supported by the presence of similar sharply demarcated and striated stains on the lower border of the saree and the sides and top of the left big toe, as would be expected had the killer's foot been placed over her own foot.

The flake of dirt detached from the left foot consisted, according to the analyst, of 2 layers; the upper (which would be nearer the skin) corresponded to the dirt found on the floor of the outer gas-stove kitchen and the lower and more external to that in the Dover-stove kitchen.

It was considered that the appreciable length of the accused's finger nails could not have inflicted the *superficial* nail marks found on the victim's neck. Unfortunately the nails of the deceased, which were of about the same length as those of the accused, did not afford any information.

Events Subsequent to the Attack

When unconsciousness rapidly supervened and the body sagged it would be natural for the killer to transport the deceased to the garage for greater privacy. The transport, in the circumstances, would most conveniently be effected with the body in the upright position, especially necessary to negotiate the narrow door-way and walled passage leading into the garage. It is probable that his hurried progress was brought to a sudden stop when the back of the deceased's body, in the position in which she was being carried, was obstructed by the lintel of the door. This deduction was made from the injuries found on her back, viz.:

1. A group of abrasions 1/16″ to ⅛″ in diameter over an area 2½″ in diameter on the back between the shoulder blades, with an underlying contusion 4¼″ × 3″; and

2. A contusion 1½″ in diameter over the middle of the left shoulder blade.

The position of the injury in the trough between the shoulder blades was of special importance, as it could have only been caused by impact with a hard narrow projection such as the lintel of the door. If this inference

was correct, the drag mark found on the floor of the kitchen could be attributed to the sliding of the killer's foot when his progress was in this way obstructed. The body, while probably still alive, was then laid on the floor of the garage.

The events which probably followed were:

1. That the killer had sexual intercourse with the deceased.
2. Finding that the deceased was still alive he had hit her with the billet of firewood found near the body.
3. That he had fetched the hand-mirror from the bedroom with the probable intention of testing for respiration.
4. That finding that she was not dead, he had placed the mortar over the chest of the deceased and stood on it.

The Question of Sexual Intercourse. A sexual assault was suggested by the posture and disposal of the clothes on the body and this was supported by the finding of a large quantity of seminal fluid in the vagina. Heavy seminal stains on the petticoat, in addition to similar stains on the bed sheet and a 'napkin' found in the bedroom were, besides, reported by the analyst. The accused, however, admitted intercourse with his wife that morning. The question of a subsequent sexual episode, inferred only from the quantity of seminal discharge, had therefore to be left a matter of doubt, especially as the grouping of the seminal stains did not yield more conclusive information.

The Blow with the Billet of Firewood. This was suggested by a contusion (6 inches long, one inch wide in front and 2¼ inches at the rear end) on the right side of head, extending from the outer angle of the right eye to 3½ inches behind and involving the upper half of the right ear.

The journey upstairs can only be surmised from the presence of the fragment of firewood found on the front stairs and the lady's hand-mirror in the garage. Unfortunately no finger-prints were found on the mirror.

The role of the mortar was inferred from its original position on the victim's neck and the foot print on its side. Here too, unfortunately, no worthwhile evidence in the way of identification of the foot print was available.

Opinion of Sir Sydney Smith

In view of the disparity between the position adopted by the Prosecution, supported as it was by its medical witnesses, and our own reconstruction, it was most fortunate that Sir Sydney Smith should have consented to give evidence for the Defence and to have made himself available as a witness at the trial.

Sir Sydney's opinion about the time of death, based on the experiments carried out on the dead bodies of the 3 criminals, were of considerable importance in his conclusions. The salient points on which he drew his inference are shown in Table 2.

Table 2.

	Temperature at 11 a.m.	*Temperature at 7 p.m.*	*Temperature Fall*	*Average Fall per Hour*
Case No. 1	96·5° F.	90·0° F.	6·5° F.	0·8° F.
Case No. 2	96·8° F.	90·4° F.	6·4° F.	0·8° F.
Case No. 3	97·5° F.	91·4° F.	6·1° F.	0·76° F.

On the basis of these observations, carried out on the dead bodies of 'full-blooded men', he considered that it would be safe to assume a rate of fall of 0.8°F. per hour in the case of Mrs. X, and that a rate of 0.7°F. per hour 'would be all right for long periods such as 20 hours or so, but it is not enough for short periods'.

He fixed the time of death as around 11 a.m. and said, 'I feel in my own mind that she did not die before 10.30 a.m.'.

With regard to the manner of the killing, Sir Sydney laid great importance on the injuries on the back of the deceased and had in fact, when he visited the scene soon after his arrival in Ceylon, paid special attention to the door-jamb in the Dover-stove kitchen. He considered it probable that the deceased had been dealt a blow on the side of the head from behind with the billet of firewood when she entered the Dover-stove kitchen. She had retreated from her attacker, perhaps in dazed condition, and had then been strangled by a left-hand grip with her back against the door-jamb, the killer standing on her left and slightly behind. He considered that the lower marks in wound No. 4 with the concavities to the right were produced with her own hand when she had attempted to free herself. The numerous marks on the neck may have been produced when the killer shifted his grip or when he used both hands for the purpose. 'To have a second assailant would be an unnecessary luxury in the case.' The drag mark, according to Sir Sydney Smith, may possibly have been caused by her foot when she was dragged from the kitchen into the garage after the strangling. He considered that sexual intercourse subsequent to the attack had to be left in doubt, owing to insufficient confirmatory evidence.

The case was tried by Mr. Justice Gratiaen and extended over a period of almost 2 months. Among the more important facts which probably aided the jury in their verdict reference may be made to the following:

1. *The Time of Death.* The consensus of medical opinion on which the Prosecution relied fixed the limits of the time of death as between 8.15 a.m. and 11.30 a.m., with no definite restriction to a time before 9.30 a.m. Our evidence, supported by that of Sir Sydney, based as it was principally on our experiments, excluded a time earlier than 10.30 a.m. (when the accused himself had left the house).

It was most unfortunate, however, as was commented on by the judge, that the time of delivery of a letter, dated the previous day and found

in the deceased's handbag, had not been investigated by the Police Inspector in charge of the case.

2. *The Manner of the Killing.* The two surgeons and Dr. Taylor were of opinion that the strangling had been effected from in front, the former favouring a right-hand and Dr. Taylor a left-hand grip. They considered that the all-important injuries between the shoulder blades could have been effected as readily on the bedroom floor as against a wall. They favoured the floor owing to the absence of any lime from the wall on the clothes of the deceased. It was unfortunate, as was remarked by the judge, that none of these prosecution witnesses had visited the locus, which would, no doubt, have helped them in coming to a more definite decision. Sir Sydney definitely excluded the bedroom floor in the causation of the injuries between the shoulder blades on account of the associated contusion. His interpretation of this injury was that it had been inflicted when the deceased retreated against the door-jamb in the kitchen, where she was strangled, the assailant being then on her left and slightly behind her.

Our evidence envisaged the assailant operating from behind, as shown in Fig. 4. We attributed the injuries to the deceased's back coming in contact with the lintel of the door (the door-way, it will be remembered, was only 18 inches wide) when she was being carried unconscious from the kitchen into the garage.

Fig. 4. The manner of the strangling as demonstrated by the writer.

The fact of the kitchen dirt adherent to the feet of the deceased and the drag mark on the kitchen floor were construed by Sir Sydney and by us as proof that the strangling had occurred in the kitchen while the deceased was standing. This was discounted by the Prosecution, who maintained that the accused had daubed the feet of the deceased with kitchen dirt in order to further fix the murder on the servant. The Government Analyst supported our statement that after the flake of dirt had been detached from the sole of the left foot, the foot still showed the dark stain of the kitchen dirt. This evidence, together with the results of his analysis of the flake as being composed of dirt from *both* kitchens, helped to disprove the Prosecution's

story, and further established that the deceased had walked barefoot (as is usual in this country) through the gas-stove kitchen into the Dover-stove kitchen and had never walked out.

The Finding of the Pearl in the Garage. It would appear that the finding of the pearl in the garage indicated that the attack had occurred in greater proximity to the garage, viz. in the kitchen rather than in the bedroom upstairs for, if it had been detached in the bedroom as a result of the attack, it would have had to survive the jostling of the body in its transport down the stairs and through the various rooms of the house.

The Finding of Garlic in the Froth in the Wind-Pipe. This has not been referred to before, but the evidence of the ayah and the subsequent police investigation both established that garlic juice was found only in the kitchen. This rather indicated, when correlated with the other evidence, that the deceased had entered the kitchen and consumed the garlic there.

Reference must also be made to the Defence assertion that the servant was the sole culprit. The Prosecution asserted that the nail marks found on him, admittedly produced by the nails of the deceased, were inflicted while the servant was gripping the deceased's hips. No injuries were found around the hips to support this theory. Besides, it would be inconceivable for the deceased to have completely 'by-passed' the strangler, operating on her neck, to reach the accomplice even if she had been partially unconscious and acting 'semi-purposively'.

The jury returned a unanimous verdict of 'not guilty' and the accused was acquitted. Three of the Crown witnesses who testified to seeing of the servant round about 10 a.m. on the day of the murder were convicted of perjury.

Brain Injuries as a Cause of Oesophago-Gastro-Duodenal Ulceration*

II. Ulcer and Brain: A Post-Mortem Study

Jørgen B. Dalgaard, M.D.

The traumatic origin of some cases of oesophago-gastro-duodenal ulceration is threefold:

1. *Direct blunt injuries to the thoraco-abdominal region* may cause mucosal slits or real ruptures, usually associated with immediate and dramatic symptoms which leave no doubt about the traumatic origin.

2. *Direct trauma* may be followed after an interval of varying length by symptoms and signs of chronic peptic ulceration. Such traumatic cases are rare and will usually be rejected by the insurance boards.

3. Finally, *traumatic brain injuries* may cause indirect (neurogenic) ulcerations. Such cases are not rare but fairly little is known about them. They usually occur soon after the cerebral injury and are, in most cases, first encountered on the autopsy table. They may be a contributory or the main cause of death. A few instances may possibly develop into chronic peptic ulcers. Cases of this last-mentioned category are the object of the present paper.

Previous Statements

The first connexion between head injury and oesophago-gastro-duodenal ulceration was noted in 1772 by John Hunter, who observed gastromalacia in a man whose skull had been fractured by a blow from a poker. Hunter did not use the word gastromalacia, but his clear description of it under the title *On the Digestion of the Stomach after Death* is unsurpassed.

Harvey Cushing's Balfour Lecture in 1932 on *Peptic Ulcer and the Interbrain*, is the classical paper on the connexion between brain lesions and acute gastric ulcer and malacia. But Cushing's cases followed cerebral operations and vascular lesions, and will not be discussed here as they were not traumatic in the sense we are discussing the problem.

* *From the* Department of Pathology, University of Aarhus, Denmark (Professor W Munck, M.D.), The University Institute of Legal Medicine, Copenhagen, Denmark (Professor K. Sand, M.D.) and The Gade Institute, Department of Pathology, University of Bergen, Norway (Professor E. Waaler, M.D.).
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From a pathological and pathogenetic point of view there is, of course, no important difference between accidental and other cases, but from a forensic point of view there certainly is.

Four cases of head injuries with acute oesophago-gastric ulcerations were observed by Opper and Zimmerman (1938) in a series of 22 ulcer cases. Their detailed report included histological examination proving the recent intravital nature of the ulcerations.

Saar (1941) encountered 7 cases of acute gastro-duodenal ulcers, 4 of which caused death by perforation, among 81 autopsies performed upon cranio-cerebral war casualties. Acid softening of the stomach was noted in most cases. Unfortunately, the interval from the skull injury to the occurrence of ulcer symptoms or death was not stated, but it appeared to be fairly long. On the basis of his observation of an ulcer frequency 3 times the average and in view of the clinical and experimental findings of others, Saar considered the connexion between brain injury and (acute) gastro-duodenal ulcer as obvious and important from a forensic point of view. The presence of bridging symptoms should not be necessary for accepting a causal relation, according to Saar.

The important investigation of Strassmann (1947) was based upon the hitherto largest single series of cases. Some of them, however, displayed only minor gastrointestinal lesions. But 80 cases would fit the definitions of acute oesophago-gastro-duodenal ulceration used here and were, with only a few exceptions, associated with intracranial lesions. Sixteen of these were injuries with multiple gastric erosions in one case, and perforating gastromalacia in 15 cases.

Wyatt and Khoo (1949) reported 2 traumatic and 2 non-traumatic cases of acute oesophago-gastro-duodenal ulceration observed among 210 instances of fatal war head injuries and coroner's cases. A post-mortem origin was excluded by the demonstration of convincing vital signs and because the autopsies were performed shortly after death.

Fletcher and Harkins (1954) in a brief review of 42 cases of acute peptic ulcers observed among 4,102 autopsies, included one head injury.

Risholm (1956) reporting 5 cases of acute upper alimentary tract ulceration, mentioned a head injury with both acute gastric erosions and a chronic duodenal ulcer.

Most reported cases concern acute ulcerations. Ask-Upmark (1939), however, reported a case of chronic gastric ulcer subsequent to a head injury in a 49-year-old man. Melaena and epigastric pains occurred shortly after the accident. Two years later a niche was demonstrated and gastric resection performed. In spite of obvious bridging symptoms an application for compensation was eventually refused by the insurance board.

The Viennese material of Bsteh (1951) contained several neurogenic ulcer cases, among which one occurred after a head injury. The age was

not stated. There had been a compound frontal fracture. Two years later ulcer symptoms developed and 3 years after that gastric resection was performed for a duodenal ulcer. Death occurred from post-traumatic meningitis one month later. The ulcer was considered a consequence of the accident 5 years before.

The present author has investigated the connexion between oesophago-gastro-duodenal ulceration and cerebral lesion in a wide sense. (A fuller report will be published in detail elsewhere.) The basic material consisted of 3,000 hospital and 1,317 medico-legal autopsies, in 385 of which ulcerations were encountered. These were divided into the following types:

(*a*) *Oesophago-* or (*b*) *Gastro-malacias*, which are ragged perforations of discoloured, flimsy and friable areas of the gastric fundus (84 cases) or the lower end of the oesophagus (41 cases), usually perforating into the left pleural cavity.

(*c*) *Haemorrhagic erosions* (51 cases), usually multiple, but limited to the mucosal membrane.

(*d*) Real *acute ulcers* in the stomach (24 cases) or (*e*) in the duodenum (42 cases) penetrating the submucosa or muscle layer, sometimes even perforating. Ordinary *chronic peptic gastric or duodenal ulcers* (88 cases in (*d*) and 65 cases in (*e*)) and *cases with gastric resection performed for ulcer* (43 cases (*f*)) were also counted out. Several cases displayed more than one type of gastro-duodenal ulceration.

The most important result was, that with only two exceptions, *acute ulceration was only found in cases with cerebral lesions, acute intoxication or acute severe stress*. Of the chronic peptic ulcer cases, 38% revealed such findings. The corresponding percentage in the complete basic material was 25%.

The types of brain lesion encountered in the 208 cases with acute and 177 cases with chronic ulceration (figures for the latter in brackets) were the following:

Traumatic brain lesions: 35 (+ 6).
Brain operations: 23.
Vascular brain lesions: 67 (+ 33).
Brain tumours: 15 (+ 6).
Intracranial infections: 11.
Intoxications: 20 (+ 8).
Burns (one case of freezing) 4 (+ 1).
Cerebral hypoxia: 9.
Severe acute stress: 15.
Other cerebral diseases: 7 (+ 12).
No cerebral lesion found: 2 (+ 71).
Brain not examined: 0 (+ 40).

Cases which were ascribable to more than one group (i.e. tumour and operation, trauma and bleeding) were classified according to the main acute lesion. For details the reader is referred to the other papers.

The Present Investigation

Of the above-mentioned 4,317 autopsies, 35 cases with acute oesophago-gastro-duodenal ulceration and 6 with chronic ulcers were associated with *traumatic brain lesions*. One case with a subacute gastric ulcer was later added. These 42 cases are the basis of the present study. In what follows acute and chronic cases will be considered separately. In each group a few outstanding cases will be reported in detail, the remaining cases being briefly surveyed in Tables 1 and 2 (Appendix).

Report of Cases

(*a*) Cases with Acute Ulcerations

Case 90. A 24-year-old man, involved in a traffic accident, sustained a severe skull injury and was admitted in an unconscious state. Extensive bilateral fractures of the cranial vault were seen on the radiographs. The spinal fluid was haemorrhagic. A small subdural haematoma was evacuated without relief of the symptoms. The patient had several haematemeses. He remained unconscious until death 3 days and 12 hours after the accident.

Autopsy. An extensive but thin subdural haematoma covered the left hemisphere. The frontal and temporal lobes displayed a very large contusion (Fig. 1) extending to the basal ganglia. The left parietal lobe showed minor contusions and there was a pronounced cerebral oedema.

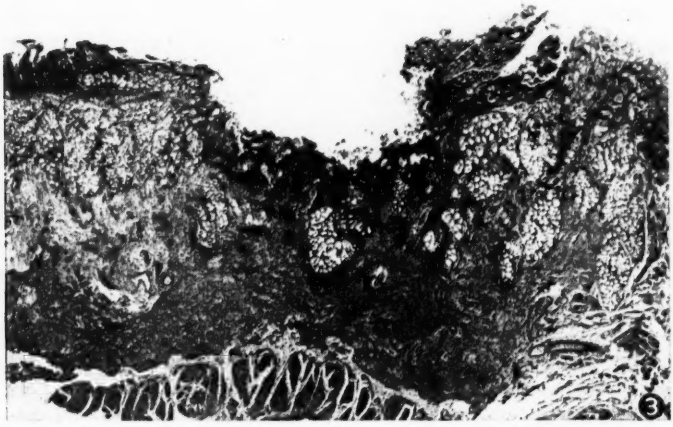
The left pleural cavity contained blood-stained acid gastric fluid, which also surrounded the fundus of the stomach. A large and a small perforation due to oesophagomalacia were found. A gastromalacia had perforated the left dome of the diaphragm (Fig. 2). Three sharply demarcated haemorrhagic erosions were noted in the first part of the duodenum. Histological examination revealed acute haemorrhagic oedema with ulceration of the mucosa and slight leucocytic infiltration but no fibrosis or vascular lesions indicative of chronic ulceration (Fig. 3).

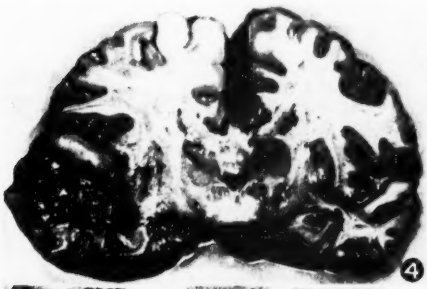
Case 338. A 65-year-old, previously healthy man had been involved in a traffic accident and was admitted in a semi-comatose state with a right frontal fracture. The following day he had haematemeses. A small subdural haematoma was evacuated from the left side without benefit. Death ensued on the sixth post-traumatic day.

The autopsy (64 hours after death) disclosed a right fronto-temporal fracture with slight depression, an extensive contusion of the temporal lobe with a 6 cm. haematoma in the right hemisphere and pronounced cerebral oedema (Fig. 4). In the duodenum a crescent-shaped, sharply demarcated ulcer (2 × 1 cm.), with a haemorrhagic floor, and a second

Figs. 1–3: *Case* 90 (*See text*).
Fig. 1. Large fronto-temporal brain injury.
Fig. 2. Two perforating oesophagomalacias and one gastromalacia.
Fig. 3. Acute duodenal erosion with necrosis and oedema but no lymphocytic infiltration or fibrosis indicative of chronic lesion. (Haematoxylin and eosin. × 14.)

minor ulcer were found (Fig. 5). The intestine contained plenty of blood. Histological examination of the duodenal ulcer revealed an acute haemorrhagic necrosis with leucocytes but no fibrosis (Fig. 6).





Figs. 4–6: *Case* 338. (*See text.*)
Fig. 4. Large injury in right hemisphere.
Fig. 5. Crescent shaped, acute duodenal ulcer.
Fig. 6. Photomicrograph from the margin of the ulcer, showing haemorrhagic oedema and recent necrosis but no evidence of chronic ulcer. (H. and E. × 90.)

(*b*) Cases with Chronic Ulcers

Case 248. A male, born in 1894, had malaria in 1912 and a soft chancre in 1922. No luetic infection was admitted, but positive sero-reactions were found in 1948 and antiluetic treatment was given. Slight dyspeptic symptoms were mentioned first in May 1941 and reappeared occasionally during the following two years. In September 1943 hunger pains induced a visit to the doctor. A barium meal was entirely negative. A light diet was prescribed.

In December 1944 the patient fell from the fourth floor and sustained a cerebral concussion with unconsciousness for 5 hours and afterwards amnesia and headache. There were no signs of skull fracture but 3 ribs and a metatarsal bone were broken. However, a rapid improvement followed and on the fifth day he was out of bed.

He now complained of severe epigastric pain and dyspepsia. Achylia was demonstrated and hydrochloric acid prescribed. But from now on he suffered

Table 1

No. *Sex* *Age*	*Cerebral Lesion* *Main Course* *Survival Time*	*Oesophago-Gastro-Duodenal Lesion* (*Time before autopsy*)	*Group*
7 M 56	Basal fracture, cortical lesions of both frontal and left temporal poles. Subarachnoid haemorrhage. Survived 4 days.	15 mm. acute duodenal ulcer. (33 hours). Necrotic ulcer with haemorrhage and leucocytes. No fibrosis.	(e)
18 M 49	Suicidal right temporal shot through both frontal lobes. Unconscious. Debridement. Survived 47 hours.	8 × 6 mm. superficial duodenal ulceration; gastromalacia not yet perforated. Acute haemorrhagic erosion (20 hours).	(e) (b)
34 M 31	Compound right temporal fracture with extensive brain lesion. Debridement. Survived 51 hours.	Oesophagomalacia perforating into left pleura. Also a 3 cm. gastromalacia. (43 hours).	(a) (b)
35 M 74	Right parietal injury causing a large contralateral subarachnoidal bleeding with unconsciousness and right hemiparesis. Survived 55 hours.	Large ragged trans-diaphragmatic gastromalacia. *Histologically:* Hyperaemia, no cellular infiltration. Several erosions and acute ulcers in upper 12 cm. of the duodenum, the largest 1 cm., nearly perforating. Haemorrhagic duodenal contents. (8 hours.)	(b) (e)
36 M 61	Ran into a train. Compound frontal fracture with extensive brain lesion. Costal and scapular fractures. Survived 63 hours.	Gastromalacia perforating at autopsy, no vital lesions. Duodenal mucosal haemorrhage and a recent perforation into the pancreas. No peritonitis. *Histologically:* Acute perforating ulcer with hyperaemia, lympho- and leucocytes. No fibrosis. (33 hours.)	(b) (e)
54 M 68	Basal fractures, bleeding from right ear. Frontal subdural haematoma; right frontal and cerebellar lesions. Unconscious, later semi-comatose and febrile. Survived 7 days.	Perforating oesophagomalacia; 100 ml. blood stained gastric acid in left pleural cavity. *Histologically:* Vital lesions (hyperaemia and leucocytic infiltration) in perforation margin and visceral pleura. (3 days.)	(a)
69 M 56	Head injury during inebriation. Next day confused and hemiparetic. Left parieto-temporal fracture, extensive cortical lesions, cerebral herniation and bleeding in the pons. Survived 8 days.	Gastromalacia perforating at autopsy (60 hours).	(b)
90 M 24	See text and Figs. 1–3.	(4 days.)	(a) (b) (e)
91 M 46	Peat-fork through right orbital roof and frontal lobe, into the left basal ganglia. Unconscious, febrile. Survived 6 days.	Gastromalacia with 4 perforations. Numerous petechiae in duodenal mucosa (60 hours).	(b)

No. *Sex* *Age*	*Cerebral Lesion* *Main Course* *Survival Time*	*Oesophago-Gastro-Duodenal Lesion* *(Time before autopsy)*	*Group*
181 M 27	Head injury. No fracture, but extensive cortical and subcortical lesions, mainly of right hemisphere. Unconscious. Also femoral fracture and pneumonia. Survived 2 days.	Gastric mucosal haemorrhages and 3 acute ulcers, up to 4 mm. in size, in pylorus and duodenum (24 hours)	(d) (e)
209 F 86	Senile and hypertensive patient fell and had a frontal injury without fracture. Thereafter stuporose with vomiting. Survived 4 days. Cerebral hyperaemia and vascular degenerative lesions.	A 2 cm. ulcer on the lesser curvature and several gastric erosions. *Histologically:* Acute necrotic ulcer with leuco- and lymphocytes, but no fibrosis (17 hours). Fig. 7.	(c)
211 M 6	Traffic accident. A bicycle handle was forced into the mouth. Carotid artery impairment. Progressing hemiparesis and coma due to cerebral injury. Minor adrenal haemorrhage. Survived 4 days.	Perforating gastromalacia and incipient oesophagomalacia without perforation (8 hours).	(b) (a)
308 M 56	Hit the occiput during an epileptic seizure. No fracture. A large subdural haematoma evacuated on the tenth day without much relief. Large melaena on 17th and 24th days. Extensive bilateral cerebral contusions in both hemispheres revealed at autopsy. Survived 27 days.	A recent ulcer, 8 mm. in size and 2–3 mm. deep, on the greater curvature of the stomach had given origin to a large intestinal bleeding which was considered a contributory cause of death. No histological examination (3 days).	(d)
309 M 73	Left temporal fracture with underlying subdural haematoma and extensive bilateral cerebral contusions. Costal fractures. Survived 5 days.	1·5 cm. duodenal ulcer just below the pylorus. No cicatricial retraction (26 hours).	(e)
310 M 53	Large fracture on the right side with subdural haematoma and contusion of both frontal lobes. Survived 36 hours.	Gastromalacia (12 hours).	(b)
311 M 74	Concussion without fracture. Death in pneumonia on seventh day. Autopsy revealed cerebral oedema, hyperaemia and pronounced atheromatosis.	Gastromalacia perforating at autopsy (21 hours).	(b)
312 M 51	Found dying. Course of events unknown. Basal fracture, subdural haematoma and extensive cerebral contusions on right side, presumably some days old. Cerebral herniation and bleeding in the pons.	Oesophagomalacia with a 4 cm. perforation into the left pleural cavity, which contained 450 ml. gastric fluid (7 hours).	(a)

No. *Sex* *Age*	*Cerebral Lesion* *Main Course* *Survival Time*	*Oesophago-Gastro-Duodenal Lesion* *(Time before autopsy)*	*Group*
314 M 61	Left temporal fracture. Subarachnoid haemorrhage and extensive cerebral contusions. Stuporous. Haematemesis on 2nd and 4th days. Also costal fractures and haemothorax. Survived 6 days.	Numerous petechiae and haemorrhagic erosions in gastric mucosa. Blood stained intestinal contents (2 days).	(c)
315 M 48	Skull trauma. No fracture, but unconsciousness, cramps and fever. Bilateral epi- and subdural haematomata evacuated on 9th day. Bilateral temporal lobe contusion. Survived 10 days.	Pyloric ulcer, 8 mm. in size, with indication of surrounding mucosal retraction. No histological examination (45 hours).	(d)
318 M 74	Fractures of the frontal bone, ribs and pelvis. Semi-conscious for a few days. Sudden death in pulmonary embolism. Subdural, partly organized haematoma. Survived 1 month.	Just above the pylorus a slightly depressed area of 1 cm. indicated a recent (?) ulcer in the state of healing. No histological examination (4 days).	(d)
319 M 68	No fracture, but right subdural haematoma and extensive contusions of right parietal, both temporal lobes and the pons. Six costal fractures and bilateral haemothorax. Survived 16 hours.	Large gastromalacia with 9 × 8 cm. ragged perforation traversing the diaphragm (24 hours).	(b)
336 M 40	Unconscious after skull trauma without fracture. Bilateral subdural haematomata, extensive right cortical lesions and bleeding in the pons. Survived 8 hours.	Gastromalacia perforating at autopsy (30 hours).	(b)
337 M 68	Alcoholic addict found inebriated with cranial fracture. Very extensive cerebral contusion, mainly in right hemisphere, but also in the pons. Survived 16 hours.	Gastromalacia perforating at autopsy; 15 mm. shallow pyloric ulcer. No histological examination (20 hours).	(b) (d)
338 M 65	See text and Figs. 4–6.		(e)
339 M 34	Head injury. No fracture, but subarachnoid haemorrhage and extensive cortical lesions on right side. Survived 9 hours.	Peasized superficial pyloro-duodenal ulcer (2 days).	(e)
341 M 10	Left temporal fracture with small epidural haematoma. Clavicular and pelvic fractures. Survived 50 hours. Slight hydrocephalus due to 2 cm. pinealoma compressing the aqueduct.	Large gastromalacia perforating the diaphragm (23 hours).	(b)
342 M 31	Extensive right temporal and basal fractures with large epidural haematoma compressing brain. Survived 25 hours.	Large gastromalacia with early perforation (40 hours).	(b)

No. *Sex* *Age*	*Cerebral Lesion* *Main Course* *Survival Time*	*Oesophago-Gastro-Duodenal Lesion* (*Time before autopsy*)	*Group*
344 F 9	Head injury. No fractures, but extensive bilateral cortical lesions. Survived 18 hours.	Gastromalacia 'the size of a child's hand' on the greater curvature adjacent the cardia. (23 hours).	(b)
345 M 44	Head injury. No fracture, but several minor cerebral and subarachnoid haemorrhages, pronounced oedema. Survived 12 hours.	Oesophagomalacia with a 2 cm. ragged perforation into the left pleural cavity (62 hours).	(a)
347 M 65	Head injury. No fracture, but subarachnoid haemorrhage on the right side and minor bleeding in the roof of the third ventricle. Survived 5 days.	Large ragged gastromalacia (39 hours).	(b)
349 M 62	Headache preceding injury followed by unconsciousness and hemiparesis. Large right subdural haematoma evacuated, but death on the third day. Recent and old subdural haematoma, hydrocephalus and recent lesions in both frontal lobes.	Several superficial erosions up to 1·5 cm. in size. *Histologically:* Hyperaemia, lymphocytic infiltration and mucosal defects without fibrosis (48 hours).	(c)
350 M 55	Fall with costal, left temporal and basal fractures. Extensive bilateral cerebral lesions. Large epidural haematoma evacuated. Unconscious until death on the seventh day.	Transversely placed ulcer 3 cm. long on the middle of the lesser curvature. No bleeding. No histological examination (32 hours).	(d)
352 M 62	Right parieto-temporal and basal fractures with large contusion mainly of the left temporal lobe. Unconscious. Survived 64 hours.	'In the pylorus an ulcer scar.' (Examined by another pathologist.) No histological examination (53 hours).	(d)
353 M 66	Found unconscious on the road with biparietal fractures. Large contusions of the right frontal and both temporal lobes. Survived 6 days.	Gastromalacia perforating at autopsy. Several recent duodenal erosions (52 hours). Said to have suffered from peptic ulcer of which, however, no evidence was found.	(b) (e)
354 M 11	Compound frontal fracture with depression, replaced at operation. Frontal lobe lesions and minor haemorrhages in the basal ganglia and pons. Unconsciousness, haematemesis. Survived 19 hours.	Numerous erosions in gastric mucosa. Contents blood-stained (51 hours). *Histological Examination:* Acute haemorrhagic erosions.	(c)

from periodic dyspepsia of increasing intensity in spite of his dietetic régime. Four periods were described as severe. Achylia was demonstrated again in 1948, not resistant to histamine.

Radiographically a few radiating mucosal folds possibly indicated an ulcer scar on the middle of the lesser curvature. In April 1952, however,

a large niche was visible high on the lesser curvature and could now be recognized even on the radiograms of 1948 (Fig. 8). The ulcer increased in spite of dietetic measures and was ultimately suspected of malignancy.

In 1953 a high gastric resection was performed. The ulcer was 4 × 1 cm. in size and 5-10 mm. deep with fibrous margins. Histological examination revealed chronic inflammation and fibrous induration without malignancy. Post-operatively the gastric symptoms disappeared.

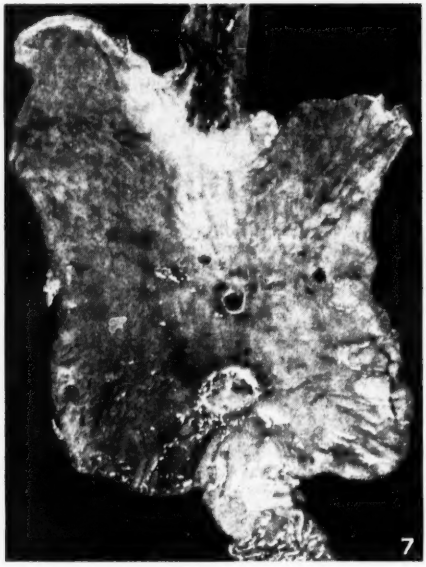
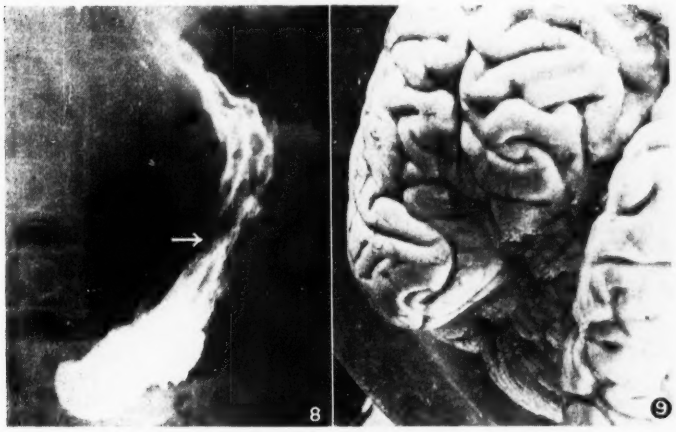


Fig. 7: Case 209 (*Table* 2). Haemorrhagic erosions and acute ulcer in the stomach of an old man who died 4 days after a head injury.

Sixteen months later rapidly progressing cardiac failure led to death. The autopsy revealed a small bronchial carcinoma with lymph node metastases invading the pericardium as the cause of death. Indications of luetic aortitis were present.

The dura displayed a greyish thickening adherent to the posterior part of the convexity of the left hemisphere. Beneath this, a depressed area (5 cm. in diameter and 5-10 mm. deep) surrounded the left calcarine fissure. The gyri were slightly atrophic here (Fig. 9). No further evidence of any cerebral lesion was found. Histological examination of the atrophic region showed degenerative cellular alterations with slight gliosis and a little pigment which was negative for iron.



Figs. 8–9: *Case* 248 (*See text*).
Fig. 8. Roentgenogram in 1948, three years after a head injury. A niche is visible high on the lesser curvature. This ulcer increased in size and led to a gastric resection in 1952. A few mucosal foldings may possibly indicate a scar lower down on the lesser curvature.
Fig. 9. A depressed atrophic area in the left cerebral hemisphere, revealed at autopsy. This is presumably an indication of the old head injury.

Case 386. An 80-year-old, previously healthy farmer had suffered for 8 years from minor cerebral insults with transient left hemiparesis, mental deficiency and dizziness. Ultimately he fell and sustained a basal fracture with bleeding from the right ear, unconsciousness and left-sided convulsions. The condition at first improved a little, but an arteriosclerotic heart lesion with arrhythmia was present, gangrene of the right foot supervened and death ensued 3 months after the accident. No gastro-intestinal symptoms had been mentioned. The sero-reactions were positive for syphilis.

The autopsy (14 hours post mortem) disclosed atrophic organs, a dilated heart with coronary atheromatosis, occlusion of the right popliteal artery but not much affection of the cerebral arteries. A thin subdural haematoma was found anterolaterally on the right side. The brain displayed several cortical lesions with yellow-brown necrotic areas, notably at the right base (Fig. 10). No lesions were seen in the deeper part of the brain. The upper half of the stomach was moderately dilated due to contraction around an ulcer on the lesser curvature. The ulcer was 8×10 mm. in size and 2-3 mm. deep with a well defined border and slight surrounding mucosal atrophy. No marked induration or thickening of the wall was present (Fig. 11).

Histological Examination. The cortical cerebral lesions revealed a disintegrated structure with numerous, large phagocytosing lipoid-macrophages, but no leucocytic infiltration. Organized microthromboses were

Figs. 10–12: *Case* 386 (*See text*)
Fig. 10. Two cortical lesions at the base of the right hemisphere.
Fig. 11. A subchronic gastric ulcer causing mucosal retraction.
Fig. 12. A photomicrograph of the shallow ulcer which was near perforation (H. and E. × 30).

seen in the superficial vessels. The findings were consistent with reactions following a lesion a few weeks old. The ulcer penetrated into the subserous layer in which some arteries were seen, surrounded by slight fibrosis. No necrotic or reactive zones and only a very slight inflammatory reaction were present. The findings were suggestive of a subchronic ulcer (Fig. 12) with imminent perforation.

Table 2: Chronic Ulcers

No. *Sex* *Age*	*Cerebral Lesion* *Main Course* *Survival Time*	*Oesophago-Gastro-Duodenal Lesion* *(Time before Autopsy)*	*Group*
363 M 31	Head injury. Death within a few minutes.	Ulcer scar in duodenum (19 hours).	(e) Chronic
364 M 72	Head injury. Subdural haematoma. Death from pulmonary embolism 8 days later.	Mucosal scar in pylorus; adhesions to the liver and gall bladder. No histological examination (65 hours).	(d) Chronic
367 M 79	Head injury. Cortical lesions and subarachnoid haemorrhage. Survived 36 hours.	Ulcer scar in stomach. Three years previously 'something wrong with the stomach' (51 hours).	(d) Chronic
369 M 55	Died of unrelated cause. An incidental finding was a brownish area of superficial softening at the base of the right frontal lobe. Dura adherent here. Interpreted as old injury.	Minor ulcer scar above the pylorus. Small ventricle (46 hours).	(d) Chronic
370 M 65	Head injury with subarachnoid haemorrhage and extensive cortical contusions in the left frontal lobe. Survived 17 days.	'Ulcer scar' in the duodenum (21 hours).	(e) Chronic ?
248 M 60	See text and Figs. 8–9.		(d) Chronic
386 M 80	See text and Figs. 10–12		(d) Chronic

Comments on the Chronic Ulcer Cases

Cases 363 and 367, in which the ulcers obviously antedated the injuries are without real interest for the present problem. In Case 364 the superficial mucosal lesion might well be a neurogenic ulcer already healing, but the presence of adhesions suggest a chronic ulcer. In Case 369 the pyloric scar might well be secondary to the alleged injury, but in the absence of reliable information, nothing is known about this. In Case 370 the autopsy

was performed by another pathologist and no histological examination was performed. A 12-day-old superficial ulcer might easily be taken for an 'ulcer scar', from the gross examination alone; it might, of course, have been an old ulcer unrelated to the head injury.

In Case 248 slight dyspeptic symptoms were present before the trauma, but a barium meal 3 months before the head injury was negative. Severe ulcer symptoms were first mentioned just after the accident. The demonstration of achylia and the localization, size and shape of the ulcer were all indicative of some special features of this ulcer. A mere coincidence of the injury and ulcer symptoms appears rather unlikely.

In Case 386 no ulcer symptoms were mentioned at all. Gross and microscopic findings at autopsy indicated a subchronic gastric ulcer with visible mucosal retraction but not much fibrosis. This might be a neurogenic ulcer some few months old. It appears most likely that the severe cerebral injury, from which the patient never recovered, was the etiological factor.

Discussion

As is known from the pioneer work of Hunter and Cushing and from numerous papers in recent years, oesophago-gastro-duodenal ulceration may arise as a consequence of cerebral lesions of various types. Cerebral injuries have also been mentioned, mostly as single cases included in non-traumatic series. Such cases are of outstanding interest from a forensic point of view.

Thirty-five cases with acute oesophago-gastro-duodenal ulceration, one with subacute and 6 with chronic peptic ulcers, all in association with traumatic brain lesions, are reported here. The types of ulceration encountered are shown in Table 3. In the following it seems appropriate to consider acute and chronic cases separately.

Table 3: Types of Oesophago-Gastro-Duodenal ulceration encountered in 42 Autopsies of Patients with Cerebral Injuries. Some Cases revealed 2 or even 3 Different Ulcerations.

(a)		Oesophago-malacia	6
(b)		Gastro-malacia	18
(c)		Acute haemorrhagic erosions ..	4
(d)		Acute gastric ulcers	7
(e)		Acute duodenal ulcers	10
(d)	Chronic	Chronic gastric ulcers	5
(e)	Chronic	Chronic duodenal ulcers ..	2

Most of the patients displaying *acute ulceration* at autopsy were the victims of traffic accidents. Several were admitted to neurosurgical departments and eventually came to medico-legal post-mortem examination.

Special interest was paid to cases of gastro- and oesophago-malacia. These are mostly considered merely as evidence of post-mortem autolysis without any significance; but,

as reported elsewhere, everyone of the 125 cases encountered in the basic material displayed some kind of cerebral lesion or acute stress. In several cases distinct vital lesions (haemorrhage, fibrin deposition, leucocytic infiltration, etc.) were observed in the edge of the perforation or on the adjoining pleura or peritoneum. These cavities sometimes contained large quantities of acid gastric fluid. Similar observations have also been reported by others. Perforation of the malacia may take place either before or after death, but, as realized allready by John Hunter, 'its cause arises in the living . . .'.

Only 2 of the 35 patients were females, mainly because men are more often involved in accidents and come to autopsy at the request of insurance boards. The ages of the 35 patients varied between 6 and 86 and averaged 50 years. Four children were less than 12 years old. This age distribution differs strikingly from that of chronic peptic ulcers.

With few exceptions the patients were admitted to hospitals and some history obtained, mostly from relatives. Four cases (Nos. 209, 308, 341, 349) revealed a history or post-mortem evidence of previous cerebral disease, but only in one case (No. 353) were pre-traumatic gastric symptoms reported. Considering the types and the severity of most of the lesions reported, it appears highly improbable that the ulcerations could have existed before the head injuries, whereas all the evidence strongly supports the assumption that the cerebral lesions were the cause of the ulcerations.

It is difficult to evaluate the significance of the various anatomical sites of the brain injuries. Lesions different in localization and size were encountered, although the frontal and temporal injuries were most common. Most cases displayed cortical and subcortical lesions with subarachnoid haemorrhage. Subdural or epidural haemorrhages were seen in some cases, either isolated or in combination with cerebral contusions. As the *cerebral oedema* seemed to be the only lesion common to all cases, the author considers this, and not the actual localization of the lesion, to be the most important factor. Increased intracranial pressure may exert its effect via an alleged hypothalamic centre (Cushing). The efferent impulses are probably carried by the vagus nerve, causing vascular and secretory influences on the affected mucosal membranes.

The material affords a special opportunity of studying the rapidity with which acute neurogenic ulceration may occur. The time of the head injury, of possible haematemeses and of death with the ulcer established, was known in nearly every case. The interval ranged from 9 hours to 30 days. Most patients had survived a few, on the average 5, days. Similar observations, including a few cases with still shorter intervals, were made in the remaining non-traumatic material. An acute ulcer or secretory and anatomical changes initiating a perforating malacia may clearly occur within a few hours after the cerebral lesion.

In a few cases histological examination of minor ulcers indicated that a process of healing was already taking place. This is in accordance with the results of animal experiments in which artifically produced acute ulcers may heal within a few days. It is also a common clinical experience that, following non-fatal cerebral injuries or surgical intervention in the brain,

haematemesis and melaena may occur without evidence of further gastro-intestinal disease, indicating that acute ulcers or erosions have readily healed.

In some cases the presence of a major gastro-intestinal bleeding or a perforating malacia with distinct vital lesions indicated that the ulceration had been the ultimate cause of death. Even in such cases the cerebral injury should be considered the underlying cause of death and the case classified accordingly. From an insurance point of view, the presence of ulceration of the types here concerned should be accepted as entirely due to the accident causing the brain lesion.

It should be mentioned that in none of the cases here reported was any serious gastro-intestinal lesion diagnosed before the autopsy.

The evaluation of cases of *chronic peptic ulcer* diagnosed after a severe head injury usually presents an intricate problem, simply because it may be difficult or impossible to tell whether the ulcer developed before the accident, afterwards but unrelated to it or was actually caused by the trauma.

Six cases with a history of a severe head injury and the finding at autopsy of a chronic peptic ulcer or its sequelae were encountered among the 4,317 autopsies comprising the basic material. Another recently observed case has been added. These 7 cases are reported and special comments are given.

In two cases the ulcers obviously antedated the injuries. When the remaining cases are considered individually, it appears possible or even likely that some of the ulcers were secondary to the brain lesions, although this could hardly be definitely proved in any case. However, when the cases are considered as a group it seems unlikely that there was a mere coincidence of cerebral injury and the occurrence of peptic ulcer in all cases.

To accept a case of chronic peptic ulcer as secondary to a cerebral injury, there should be a negative pre-traumatic history, at best a negative radiograph, adequate trauma and preferably distinct bridging symptoms during the interval between the accident and the demonstration of the chronic ulcer. It must be admitted, however, that even if post-traumatic ulcers were rather common, only a few of these would fulfil these requirements. Presumably real post-traumatic cases are rare, so it should be very uncommon to meet incontrovertible cases.

In Cases 248 en 386, adequate cerebral injuries were sustained. In the first case a negative pre-traumatic radiograph and definite bridging symptoms existed; in the other case a purely negative pre-traumatic history but no bridging symptoms were at hand. Nothing can definitely be proved, but it appears quite likely that both are instances of post-traumatic neurogenic ulcers, comparable to the cases of Ask-Upmark and Bsteh. However, several more cases of this type must be considered before 'chronic peptic ulcers secondary to cerebral injuries' could be acknowledged as a well-defined forensic entity.

Summary

This is the second in a series of papers on brain lesions and oesophago-gastro-duodenal ulceration; 208 acute and 177 chronic ulcer cases were encountered among 4,317 autopsies. The types of ulceration, which were previously described in detail, were: oesophago- and gastro-malacias, acute haemorrhagic erosions, acute ulcers and chronic gastric or duodenal ulcers. (The reference to the first paper is given below.)

The present paper concerns the cases with traumatic brain injuries. Thirty-five patients with acute ulcerations had sustained a severe head injury 9 hours to 30 days, on the average 5 days, before death. Only two patients were females; 4 were children. Most patients died of their cerebral lesion, but in a few cases the ulceration appeared to be a contributory cause of death, either through haemorrhage or perforation.

The causal relation between the cerebral injuries and the occurrence of the ulceration seems obvious in the acute cases; but the localization of the brain lesion appeared less important. Most cases revealed cortical contusions with subarachnoid bleeding, some showed subdural or epidural haematomata. Traumatic oedema is considered to be the most important single factor, presumably exerting its effect by means of secretory and vascular influences via the vagus nerve.

The evaluation of cases with chronic peptic ulcer diagnosed after a severe head injury is difficult. Seven cases are reported. Two were rejected, as the ulcers were pre-traumatic. In some of the remaining cases an etiological relation was possible, in two cases even very likely, although it could not definitely be proved. The general requirements for accepting chronic peptic ulcers as secondary to cerebral injuries are discussed.

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Penetrating Stab Wound of the Heart

Associated with Cerebral Embolism

H. Ranasinghe, M.B., B.S. (Ceylon)

Department of Forensic Medicine, University of Ceylon, Ceylon

It is the experience of all those conversant with forensic autopsies that penetrating wounds of the heart need not be necessarily, or even immediately, fatal. Moritz,[1] indeed, refers to some cases of through-and-through knife and bullet wounds of the heart which (on recovery) had slight, if any, evidence of functional or structural damage. In fatal cases, however, death occurring shortly after the injury often results from reflex shock, haemorrhage, haemopericardium or functional derangement of the auriculo-ventricular node or the conduction system; and at a later stage from complications such as pericarditis, mediastinitis or cerebral embolism.

The medico-legal interest in these cases centres round the period of survival and such inferences as whether the victim could have spoken, made a lucid statement or performed some volitional act after the occurrence of the injury; for these are all essential considerations in assessing the suicidal, accidental or homicidal nature of the incident.

The following case which came to autopsy is of considerable interest.

History

A young man stabbed himself in the chest immediately after fatally stabbing a young married woman in her house. The husband of the woman, who was away at the time, returned later to find the assailant lying prone and motionless over the body of his wife. The husband then dragged the assailant into the garden (a distance of not more than 20 feet) whereupon the latter took to his heels. After running about 200 yards (the distance being subsequently measured) he collapsed. While collapsing he had asked for a drink of water and was thereafter unconscious up to the time of his death.

From the evidence of witnesses, it was established that the stabbing occurred at about 8 a.m., the husband returned home about 8.45 a.m. and the man collapsed at 9 a.m. The police, arriving at 9.45 a.m., had the injured man admitted to a general surgical ward of a hospital immediately. Surgical treatment was not considered possible as his condition was very

poor. All resuscitative methods failed, the patient became breathless, his pulse (which earlier had been very rapid, regular and thready) became imperceptible and he died at 6.50 a.m. the following morning (*i.e.* about 23 hours after the incident). The weapon used was a 'Kris' knife* with a single-edged blade 5.5 inches (13.75 cm.) long, maximum width 0.75 inch (1.9 cm.) and tapering almost to a point.

Relevant Post-Mortem and Pathological Findings

The body was that of a sparely built young man, of about 20—25 years, with several stab wounds covering an area $3\frac{1}{4} \times 5$ inches (8.1×12.5 cm.) over the front of his lower chest and upper abdomen, with little external haemorrhage. Most of these stab wounds were superficial, but one of them had entered the chest $\frac{3}{4}$ inch (1.9 cm.) to the left of the midfront line on a level with the left nipple. It later proved to be the fatal wound. This wound was $\frac{3}{8}$ inch (0.9 cm.) long and the underlying rib (4th left rib) was cut vertically for $\frac{3}{8}$ inch (0.9 cm.). The injury was directed inwards, downwards and slightly to the left. It penetrated the following structures: the pericardium, the front wall of the right ventricle, the right ventricular cavity, the base of the interventricular septum and finally entered the left ventricular cavity. The aortic, pulmonary and coronary vessels were not injured. The septal wound was 1/5 inch (0.5 cm.) long and the pericardial and ventricular wounds, which were slit-like and oblique in direction, were 1 inch (2.5 cm.) and $\frac{1}{4}$ inch (0.6 cm.) long respectively. The total depth of penetration was $4\frac{1}{2}$ inches (11.3 cm.). There were a few sub-endocardial haemorrhages at the site of the septal injury and two small thrombi were adherent to it on either side of the septum. There were about 12 oz. of serum and clotted blood in the pericardial cavity and about 15 oz. of fluid blood in the left pleural cavity.

The brain showed an area of congestion 3×4 inches (7.5×10 cm.) in the distribution of the right middle cerebral artery; on microscopic examination this proved to be an infarct. The rest of the brain appeared normal. The right middle cerebral artery was blocked for $\frac{3}{4}$ inch (1.9 cm.) from a point 1 inch (2.5 cm.) distal to its origin from the internal carotid artery. This was later established microscopically as being due to an embolus and its associated retrograde thrombus. There was no evidence of any cardiovascular disease. The cause of death was cardiac tamponade.

Discussion

The salient points in the history are:

The deceased (*a*) stabbed himself in the chest;

* 'Kris' knife: A single-edged tapering knife. It has a straight blade 5—6 inches (12.5—15.0 cm.) long and is usually carried by villagers in a protective sheath round their waists.

(*b*) lay prone over the body of the dead woman for about 45 minutes;

(*c*) suddenly undertook comparatively violent physical exertion and collapsed at the end of it;

(*d*) uttered a few words while collapsing and did not speak thereafter though he lived for a further period of 22 hours.

The direction, grouping and superficial nature of the wounds surrounding the fatal injury (which itself was directed inwards, downwards and to the left) were typical of the self-inflicted knife wounds of a right-handed man. The absence of cardio-vascular disease in this person of 20—25 years of age makes it very likely that the embolism of the right middle cerebral artery was caused by a mural thrombus of the left ventricle. The sudden collapse, unconsciousness and inability of the deceased to speak at the end of his run of 200 yards was no doubt due to the occurrence of embolism. The thrombus in the heart must have formed during the preceding period when the deceased was inactive, and its detachment may have been precipitated by the sudden physical exertion. Gonzales *et al.*,[2] recording a few similar cases of stab injuries of the heart, state that mural thrombosis and cerebral embolism may occur within 24 hours of such injuries. The present case is of particular interest in that the thrombosis and embolism followed very soon after the injury.

It is important to remember that persons with heart injuries may perform certain actions before such injuries prove fatal. Mann and Brend[3] quote a case where a man, who was stabbed in the heart, lifted heavy weights the next day and died 4 days later. Gonzales *et al.* (*op. cit.*) record an instance where the victim, after being stabbed in the heart, ran across a road, up two flights of stairs into his apartment, and died after locking himself up in the bathroom. Davidson and Fiddes[4] instance a case where a man stabbed in the right ventricle walked a distance of 600 yards in about 20 minutes before collapsing. Glaister,[5] Greval,[6] Kerr[7] and Smith and Simpson[8] all refer to other similar cases. These examples and the case under discussion would indicate that when a person is found dead with a heart injury it should not be assumed that he was incapacitated from performing volitional activity.

The period of survival after a penetrating heart injury would naturally depend on the state of the conducting mechanism and on the amount of blood lost—especially the rate of loss. In the present case, the regular and rapid pulse rate would indicate that the conducting system was unaffected. With regard to the loss of blood, attention may be drawn to the following facts:

1. The ventricular wound was slit-like, oblique and valvular, thereby causing a slow leak of blood;

2. The pericardial cavity contained about 12 oz. of blood.

This quantity of blood would have been sufficient to cause cardiac arrest, which it undoubtedly did; but its accumulation would have taken some

time, as the wound in the pericardium was sufficiently large to allow a free escape of blood from the pericardial into the left pleural cavity. As a result cardiac tamponade was delayed.

Summary

It would appear, from the case reported, that opinions regarding volitional activity and the power of speech after stab injuries of the ventricles, should be based on a careful consideration of the following points:

1. The size and patency of the pericardial wound.

2. The nature of the ventricular injury—especially with regard to its size and direction (oblique or otherwise).

3. Involvement of the interventricular septum (and as a result the conducting mechanism).

4. The occurrence of complications such as thrombosis and embolism.

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Treatment of Acute Ethanol Intoxication

Theodore Koppanyi, Ph.D.

Department of Pharmacology, Georgetown University School of Medicine, Washington, D.C.

Carbon monoxide, ethyl alcohol and barbiturates rank first among the fatal poisons in the United States and, in fact, in most civilized countries. Of course, statistics on death from alcohol intoxication include resultant traffic casualties and miscellaneous accidents. However, a third of the fatalities are probably caused by the direct toxicological effect of alcohol on the central nervous system, or follow secondary aspiration or hypostatic pneumonia. Acute ethanol poisoning must therefore be considered an international problem.

Treatment of Acute Alcoholic Excitement

(Inebriation or excitement—characterized by gross gait abnormality, speech difficulties, flushed face, dilated pupils, and frequently anti-social behavior.)

I. *Sedation.* A wide variety of treatments is proposed for the alcoholic inebriate, but it is customary to treat violent patients by aliphatic depressants, such as chloral hydrate, paraldehyde and barbiturates, or by anti-anxiety agents, such as promazine and chlorpromazine, in order to bring about sleep and/or tranquilization. While, in the great majority of cases, this treatment is symptomatically successful, it is not without a slight risk in that it may convert the inebriation into anesthesia (McDonald and Koppanyi, unpublished).

In mice weighing 20 g. each, experiments on the combined effects of ethyl alcohol and chlorpromazine showed that while 5 mg. and respectively 10 mg. of chlorpromazine produced no sleep, the combined administration of 0·05 ml. of 95% ethyl alcohol (total dose), which in itself likewise produced no sleep, and 5 mg./kg. chlorpromazine produced sleep in 16 of 19 experimental animals, of which 3 died. In man, such fatalities almost always can be prevented if the alcoholic blood level is below 350 mg. per 100 c.c. and the patient had not ingested large amounts of aliphatic narcotics or tranquilizing agents along with ethyl alcohol. The administration of hypnotics, particularly of barbiturates, in acute alcoholic intoxication has the added hazard that the patient may become a barbiturate or paraldehyde addict in addition to the alcoholism.

These facts contradict the recommendations of Kieve[14] who admits that acute alcoholic intoxication is a disease entity potentially fatal. Yet he recommends intramuscular injection of paraldehyde, 0·2 g. of pentobarbital sodium, and 50 mg. of meperidine even in stuporous patients who are not 'too restless' and when such patients pass into the phase of slow awakening. Without adequate blood studies, it is easy to see that this procedure is too risky. Violent patients may be so treated for their own protection, but larger doses of aliphatic depressants and meperidine, and incidentally other narcotic analgesics, should be avoided.

A curious sedation, consisting of 26% alcohol, 2% liver extract, some thiamin, and 62% isotonic glucose, injected intravenously, is claimed by Lecoq[20] and Paterson[27] to restore to normal the disordered metabolism of the alcoholic patient. While it is possible that this treatment may be beneficial in the withdrawal phenomena, it is difficult to see how it could improve the condition of the acutely poisoned individual.

II. *Oxygen and Carbon Dioxide.* Davis and Robertson[6] and Robinson and Selesnick[30] reported striking successes in the treatment of acute alcoholic intoxication with the administration by inhalation of oxygen alone, or a mixture of 90% oxygen and 10% carbon dioxide, respectively. A number of early papers reported similar results in increasing the rate of alcohol metabolism, but later investigation has not confirmed these data. No or negligible effects of inhalation of pure oxygen on blood alcohol were found by Fleming and Reynolds[7] and, in experiments on rabbits, Barach[1] found the fatal dose of alcohol to be the same whether the animals were kept in air or in an atmosphere of oxygen. In dogs, Newman and Card[24] found that inhalation of 90% oxygen and 10% carbon dioxide for a period of 30 minutes was not effective in significantly reducing the total body alcohol. There was neither an increase in the rate of metabolism of alcohol nor a significant increase in its excretion through the lungs, as evidenced by the extremely low concentration of alcohol in the expired air. The experiments of Butler[2] are equally conclusive. The righting reflexes in 'midbrain rabbits' elicited by movements of the neck are depressed by an intraperitoneal dose of 1 c.c. of alcohol per kg. The inhalation of oxygen produced no return of this righting reflex. It seems fairly certain that oxygen and carbon dioxide are not indicated in the treatment of severe inebriation.

III. *Analeptics.* Orenstein *et al.*[26] observed that the intravenous administration of pentylenetetrazol in doses up to 5 c.c. of a 10% solution produced a marked tranquilizing effect within 10 minutes in 34 excited alcoholics. No further treatment was necessary. This clinical experiment, if confirmed, probably would constitute a safe treatment for alcoholic inebriates, since no untoward effects were noted. This observation is quite unusual, since a central stimulating agent quieted the excited alcoholic inebriates. This

phenomenon may be explained, however, in the light of findings by Koppanyi and Fazekas, who had shown that doses of pentylenetetrazol (leptazol), below convulsant levels, very frequently produce lassitude and sleep.

IV. *Insulin.* Ingested alcohol may be removed from the body either by increasing the rate of its oxidation or its excretion. Evidence of the blood-alcohol lowering effect of insulin is still controversial, but it was introduced on the assumption that it is necessary for the optimum rate of alcohol metabolism, which is about 380 c.c. per 24 hours. Galamini[8], Hirschfelder and Maxwell,[11] and Lang and Schlick[19] were unable to lower the blood alcohol level with insulin when compared with controls, and Widmark,[39] experimenting with dogs, found that insulin accelerates the rate of oxidation of alcohol only if the rate is not optimal. Once the optimal rate of oxidation within physiological limits was reached, further doses of insulin had no effect. Junge,[12] in experiments with rabbits, concluded that insulin administered during the absorption phase interfered with the normal absorption of alcohol and reduced the blood level, but when given after the peak was reached, had no further effect on blood alcohol. According to Junge,[12] insulin has a sobering action but does not affect motor disturbances. Careful studies by Meyer[22] developed that 0·3 unit of insulin per kg. is most efficacious in reducing alcohol concentration in the blood, notwithstanding the fact that others employed larger doses. He did not find an average lowering of more than 20%.

The use of insulin alone is not without dangers. Wortis *et al.*[40] called attention to the fact that alcoholics show a marked sensitivity to even relatively small doses. Hypoglycemia often has to be terminated with the administration of glucose, and the patients must be observed for signs of insulin shock, since fatalities have occurred during insulin treatment. This fact led Goldfarb *et al.*[9] to combine insulin and glucose in the treatment of acute alcoholism. They claim that there is a relation between alcohol metabolism and oxidation of glucose. Thiamin hydrochloride is very often used in conjunction with insulin and glucose[32, 34] as a necessary catalyst for the intermediate carbohydrate combustion. Clark *et al.*[4] used sodium bicarbonate and sodium citrate in a similar catalytic manner.

V. *Fructose.* Recently it was shown both in dogs and in man that fructose produces a marked decline of concentration of alcohol in the blood. Pletscher *et al.*[28] have shown that in dogs the intravenous infusion of fructose (2 g. per kg. per hour) decreased the blood values of alcohol by about 80%. At the same time, the urinary excretion of alcohol was not increased, supporting the conclusion that fructose accelerated the dismutation reaction during which alcohol is oxidized to acetaldehyde and pyruvate is reduced to lactate.[38] In 14 human volunteers, Pletscher *et al.*[29] found that when alcohol injections were followed by intravenously administered fructose, the rate of disappearance of alcohol from the blood was 70% faster than normal.

The combination of fructose and insulin was not tried, but the interesting results of these experiments have led to accelerated interest in the possibilities of this sugar in the treatment of alcoholism. It may be noted that the administration of fructose is always followed by a rapid increase of blood pyruvate. No clear-cut sobering effects from the administration of fructose have been reported as yet.

VI. *ACTH and Adrenocortical Hormones.* There is no conclusive evidence that in acute alcoholic states pituitary or adrenocortical deficiency exists although so theorized by Werner and Pearson,[37] and it is likely that these hormones act only as very expensive sedatives. Adrenal cortical extract (ACE) has a more prompt sedative effect than ACTH and it restores the appetite more quickly. ACE is probably preferable in female alcoholics, who occasionally do not respond to ACTH but are promptly sedated by ACE.[33] Occasionally, large amounts of vitamin C are given to supplement the effects of these hormones.[13]

VII. *Succinic Acid.* Castex *et al.*,[3] experimenting in man and animals, reported what appeared to be quite effective results in reducing alcohol blood levels by injecting succinic acid, on the theory that an enzymatic process of normal ethyl alcohol oxidation would be accelerated. However, investigations by Corson, Koppanyi and Vivino[5] have shown that succinic acid is not effective in the treatment of barbiturate poisoning. It is possible that succinate irritates the intima of blood vessels and thus produces analeptic-like effects by reflex stimulation.

Treatment of Acute Alcoholic Coma

(Anesthesia or coma—characterized by pallor, areflexia, muscular relaxation, and in the later stages by shallow abdominal respiration, hypotension, and often hypothermia.)

Death due to ethyl alcohol coma is a clinical entity of major proportions. The margin of safety between the anesthetic dose and the LD_1* is so narrow as to be practically non-existent. Alcohol blood levels ranging between 350 and 475 mg. per 100 c.c. have caused death, and levels between 550 and 900 mg. per 100 c.c. are usually fatal, if untreated. One liter of 100-proof distilled liquor consumed within an hour or less produces such blood levels with resultant coma and possibly death. Several fatal acute alcoholic poisonings have been reported following the consumption within 30 minutes of 3 or more liters of wines containing from 8—11% alcohol.[21] Children show particular susceptibility to alcohol, possibly corresponding to their weight or body surface area, and from 100 to 200 c.c. of 50% alcohol may be fatal for them. They have been severely poisoned by candies containing cognac, and by inhalation of alcohol vapors.

* LD = lethal dose. LD_1= 1% mortality; LD_{50}= 50% mortality; LD_{99}= almost absolute mortality in a given population.

The cause of death by alcohol poisoning and by other aliphatic narcotics is identical, with the usual depression of respiration and circulation. Aspiration or hypostatic pneumonia may be the indirect cause of alcoholic death. The membranes of the brain are generally hyperemic and there might be a well-marked edema at the base of the brain, with increased intracranial pressure. Pulmonary edema has also been observed.[35] The treatment of acute alcoholic coma does not differ from that of barbiturate coma, except that insulin and glucose or fructose administration may be attempted in the former and are not effective in the latter.

I. *Symptomatic Treatment.* This is still the most frequently used treatment for acute alcoholic poisoning. It consists of bronchoscopy whenever indicated, the maintenance of a patent air-way, and administration of oxygen through the air-way whenever cyanosis is present. Frequent turning, and slapping of the thoracic wall occasionally to loosen bronchial secretions, are essential procedures. The victim should be kept in a warm environment since his body dissipates heat due to vasodilatation of the skin. Parenteral fluids are administered, and 50% glucose, or preferably other sugars, are given by vein to avoid pulmonary edema and increased intracranial pressure. In the presence of peripheral circulatory collapse, plasma expanders or whole blood are given.[17, 25] Mousel[23] recommends the administration of human serum albumin to reduce cerebral edema, and cisternal or lumbar puncture may also be performed to reduce increased cerebrospinal fluid pressure. Careful attention is given to fluid and mineral balance.

In very severe cases where respiration is maintained only by hypoxic stimulation of the chemoreceptors, the patients should *not* be placed in oxygen tents or be given oxygen through nasal catheters, because respiration may cease suddenly, due to the absence of the hypoxic stimulus. The respiration of these patients must be supported in the inspiratory phase by oxygen administered through a standard anesthesia apparatus.

Mention has already been made of the administration of from 5—10% carbon dioxide. While inhalation of such a concentration of CO_2 undoubtedly stimulates respiration of comatose patients via the chemoreceptor mechanism and the respiratory centers, this therapeutic procedure should be condemned. Due to depression of respiration, the alcoholized individual already is suffering from accumulation of CO_2, and to administer carbon dioxide further may increase by additive effects the intensity of the narcosis.

II. *Analeptics.* The unpublished results of Koppanyi and Fazekas indicate that analeptics are far less effective in alcohol than in barbiturate coma, although central stimulating drugs have been used in the former. In animals anesthetized with alcohol, Siepmann[31] found nikethamide (coramine), in fractional doses, produced stimulation of respiration and a temporary awakening effect, particularly in the stage of poisoning when the blood alcohol concentration declines. In severe alcoholic poisoning in man, Krull[18] recommended that 5 c.c. of this drug be given intravenously and

5 c.c. intramuscularly at the same time. After from 5 to 10 minutes, a second intravenous injection of 5 c.c. of nikethamide generally produced a clear-cut awakening effect. In most cases, the injection of 20 c.c. was sufficient to denarcotize his patients.

In rabbits, Werner[36] conservatively reported some evidence of stimulation after the use of nikethamide, pentylenetetrazol, picrotoxin and amphetamine, but found that none effected complete arousal of depressed animals. Hess[10] found that in rats narcotized with alcohol, pentylenetetrazol (25 mg. per kg.) produced awakening, whereas larger doses were less effective in the species. Orenstein *et al.*[26] reported that 16 patients narcotized with alcohol, when given an intravenous injection of 5 c.c. of a 10% solution of pentylenetetrazol, regained consciousness in from 5 to 30 minutes and were oriented adequately. Occasionally the dose had to be repeated.

It may be concluded that analeptics have been used with moderate success in alcoholic coma. Possibly, the intravenous injection of 5 c.c. of a 10% solution of pentylenetetrazol or a corresponding dose of nikethamide awakens animals and humans only in light alcoholic coma, and are therefore useful only as diagnostic tests to determine the depth of depression.[16] Patients who show arousal following the administration of these drugs are in no need of further analeptic therapy, but patients who show no sign of arousal at the first injection, may need additional treatment and constant nursing care.

III. *Increase of Alcohol Metabolism.* Strecker and Rivers[34] reported on comatose patients with depressed respiration and low blood pressure. These patients were given 50 units of insulin, 100 mg. of thiamin and 100 c.c. of isotonic glucose intravenously. Some of their 15 patients almost immediately began to move their limbs and mutter incoherently. About one hour later they were able to give their first names. Usually 2 hours later, the patients began to show signs of hypoglycemia and were given about 600 c.c. of orange juice. Some of them thereafter showed rapid improvement and in 4 hours after admission were able to talk coherently. On admission, the blood alcohol level of a typical patient was 340 mg. per 100 c.c. and 6 hours later, 80 mg. per 100 c.c. Obviously, these patients had a lower-than-the-usual comatose or fatal level of blood alcohol; however, as already stated, death had been reported with levels as here observed. One might venture to say that in this group, the patients had taken an LD_1, or LD_5 of ethanol, but certainly not an LD_{50}.

It remains to be seen what effects fructose alone or a combination of fructose and insulin would have on the course of acute alcoholic poisoning. In this laboratory, we are currently conducting experiments which indicate that fructose is a useful drug in alcoholic coma in rabbits receiving 6 c.c. of 95% alcohol per kg. of body weight. Thus far we have administered fructose in doses of 10 g. per kg. along with alcohol, orally, in the doses

mentioned above. This procedure significantly kept down the blood alcohol levels, and the animals did not lapse into coma. When alcohol is given by mouth, and fructose in smaller intravenous doses one hour later, the sugar appears to accelerate significantly the metabolism of alcohol and produce a rapid awakening effect from alcohol coma.

IV. *Acceleration of Alcohol Excretion.* Alcohol is distributed throughout the total body water, and is therefore normally excreted in the urine only in small percentages of between 2—5%. Koppanyi and Cutting[15] have shown that massive intravenous infusions of 1% sodium chloride in dogs saved the lives of animals receiving LD_{99} of orally administered ethanol. The animals received from 30—40% of their entire body weight in saline within 2 to 4 hours, and excreted on the average of 25% of the infused fluids during the course of the treatment. Possible duplication of the results could be made with hemodialysis by means of the Kolff-type artificial kidney. The authors' interpretation of the beneficial effect of massive intravenous infusion is that the alcohol equilibrates with additional infused fluids and the neurotoxic effects are lessened. This applies specifically to neurotoxic poisons because the central nervous system does not become hydrated as a result of massive infusions. In this connection, it should be emphasized that alcoholics, and in fact all comatose patients, should be catheterized and the rate of massive intravenous infusions regulated according to the rate of urine flow. This is necessary to avoid complications such as overloading of the circulation, pulmonary edema or possibly edema of the brain.

Summary

The treatment of acute inebriation varies. Sedative agents such as aliphatic depressants, meperidine and the recently developed anti-anxiety agents are often used but they must be administered cautiously so as to produce a restful sleep but not a state of anesthesia. Every patient in alcohol anesthesia, however light, may die because of complications.

Oxygen alone or oxygen with carbon dioxide is frequently used in treating inebriates. However, carefully conducted experiments have shown that neither oxygen alone nor oxygen and carbon dioxide reduce significantly the total body alcohol. It is therefore difficult to explain clinical reports that inhalation of the above gases simulates the effects of sedatives.

Leptazol was reported to sedate excited alcoholics within a few minutes. The probable explanation for this phenomenon, if confirmed, is the stimulation of the depressed inhibitory centers in the cerebral cortex so that the excitement, which may be due to release from cortical inhibition, is abolished.

Insulin alone and insulin and glucose have been reported to accelerate the rate of oxidation of alcohol. Glucose acts mainly as a catalyst, although other catalytic agents have also been combined with insulin and glucose.

Hypoglycemia has also an apparently sobering effect. The reports on the effects of insulin and glucose are conflicting. The conflict might be resolved if it could be shown definitely that insulin accelerates the rate of metabolism of alcohol only in cases where primary deficiency of insulin exists or when alcohol metabolism, for some unknown reason, depends on a relatively high insulin level. At any rate, insulin appears to be necessary for the optimum rate of alcohol metabolism, particularly in the liver. Once optimum rate of alcohol metabolism is obtained, it is difficult to see how insulin can further accelerate the oxidation of alcohol.

Powerful metabolic stimulants, e.g. thyroid or dinitrophenol, have no influence on the rate of oxidation of alcohol. This is more or less fixed and, in ordinary circumstances, is accelerated only slightly by increasing the amount of alcohol present in the body.

The use of fructose in the treatment of acute alcoholism is still in the experimental stage. It is clear, however, that it increases blood pyruvate and the rate of disappearance of alcohol from the blood. Possibly it also causes an accumulation of acetaldehyde. Since Antabuse also produces an accumulation of acetaldehyde, it is within the realm of possibility that fructose could be used as a less toxic substitute.

ACTH and adrenocortical hormones may produce a calming effect in acute alcoholic bouts of chronic alcoholics. Apart from their non-specific sedative action, they can be effective only in deficiency states of these particular hormones.

Acute alcoholic coma is essentially a deep anesthesia, with loss of reflexes, muscular relaxation, cold and occasionally cyanotic skin, stertorous respiration, and frequently dilated and fixed pupils.

The fatal dose of alcohol is variable: from 1—2 pints of 100-proof distilled liquor taken within a short time may produce death. Children require smaller doses. Death occurs usually within from 6—12 hours and is due to respiratory and/or circulatory failure. In some instances when the fatal outcome is delayed for 5 or 6 days, death is not directly due to alcohol but to pneumonia. If the coma persists for more than 12 hours, life is usually in danger. It is therefore imperative to terminate the coma as early as possible.

The blood level of alcohol is likewise indicative of the depth of the coma. In a few cases, 350 mg. per 100 c.c. caused death, and the higher the blood level, the more frequent the fatal outcome. When the blood level is higher than 600 mg. per 100 c.c. the patients frequently die if they are untreated.

It is unfortunate that no reliable treatment for severe alcoholic coma is available. The best remedy is probably the symptomatic treatment by good and continuous nursing care for at least 12 hours to keep the respiratory passages free, to keep the patient warm, to turn patient frequently, ad-

minister antibiotics to prevent pneumonia and, if necessary, to employ artificial respiration and/or transfusions of whole blood or plasma expanders.

Insulin and glucose and fructose may be employed to accelerate the combustion of alcohol.

Analeptics (central stimulants) should be resorted to only in conjunction with other measures. There is no conclusive proof that analeptics alone awaken patients from deep alcoholic coma and maintain them in states of normal reflex excitability and muscular tone.

Finally, since a quart of urine in deeply comatose patients may contain as much as 5 g. or more of alcohol, massive intravenous infusions of saline may be employed to accelerate the urinary excretion of ethanol. In these cases, however, one should proceed cautiously and consider the possibility of cardiac embarrassment, and of pulmonary and cerebral edema. There is also a strong possibility that hemodialysis by means of artificial kidneys may prove useful in the treatment of acute ethanol coma.

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Reviews of Books

Medical Malpractice

Malpractice Liability of Doctors and Hospitals. By William C. J. Meredith, Q.C. 1956. (Pp. 300 + viii.) Canada: The Carswell Company Limited.

The author of this volume is a distinguished Canadian silk who is Dean of the Faculty of Law at McGill University. He has also published another monograph of interest to medico-legal practitioners viz. *Insanity as a Criminal Defence.*

His current volume on liability for medical malpractice naturally covers Canadian law, but the greater part of its scope is substantially applicable also in England and the United States. Because the factual cases used to illustrate the various forensic issues are of enduring interest to medical practitioner, from the doctor's point of view the whole volume is of value, since litigation involving medical practitioners has increased in recent years to a considerable and totally unforeseen degree.

The liability of hospitals was fairly clearly defined well before the era of the Welfare State, but the adoption of socially advanced measures reflected in the provision of free hospitalization has certainly also made the hospital as an institution more vulnerable as an object of attack by the litigious patient.

The increasing extent to which the state has assumed responsibility for providing hospital services makes the chapter on *Liability of Hospitals* very instructive for full-time members of hospital staffs, medical superintendents and administrative officers. This chapter also deals with the position of interns and nurses. Of particular importance is the chapter on *Malpractice in Diagnosis and Treatment,* which deals with the important principle of a doctor's right to be wrong. There has, e.g. in recent times been considerable interest in the legal consequences of irradiation damage resulting from the treatment of disease. It is thus of interest to note that 'although a doctor is responsible for burns caused by negligent use of the apparatus, it has been held that a patient undergoing X-ray treatment assumes the risk of receiving a burn from a *proper* exposure' (p. 74).

In the first two chapters Professor Meredith traverses the fundamental relationship between doctor and patient (the Hippocratic foundation for which is of universal applicability as far as the medical profession is concerned) and the principles governing professional secrecy. These introductory chapters are followed by one on *The Doctor as a Witness* which will repay careful study by the undergraduate student as well as the established practitioner.

The physician involved in workmen's compensation work will find the chapter on *Assessment of Damages in Bodily and Fatal Injury Cases* of particular practical utility.

The section on *Abortion* throws into relief the inadequacy of the law in some parts of the world in respect of the therapeutic reasons for interrupting pregnancy, especially in view of the newer knowledge of the effects of virus infections on the foetus during the early months of pregnancy. It is here that the medical and legal approaches diverge most seriously, as a therapeutic abortion in these circumstances raises the question of a 'mercy killing' of the foetus.

Regan has defined the indications for therapeutic abortion to include not only situations when there is risk to the life or *health* of the mother, but also when the foetal hazard is markedly increased (quoted at p. 218). This attitude is not provided for in most jurisdictions and is clearly the cause of much genuine difficulty for the practitioner whose only recourse, if he is in doubt about the position, is to take certain legal precautions, e.g. that the abortion be performed openly and in a recognized hospital without any suggestion of a 'hole-in-the-corner' business about it (p. 228).

The volume is most thoroughly documented with case references and a comprehensive *Table of Cases Cited* is a feature of the book as a separate index (pp. 241-260). The *Subject Index* extends from pp. 261-300.

This excellent survey is a most necessary addition to the library of all medical and legal practitioners.

Jervis on Coroners

Jervis on The Office and Duties of Coroners. By W. B. Purchase, C.B.E., M.C., M.B., D.P.H. and H. W. Wollaston. 1957, 9th ed. (Pp. 554 + Index. 84s.) London: Sweet & Maxwell Ltd.

With its present publication this standard work reaches its 9th edition under the authorship of Dr. W. B. Purchase (Coroner to Her Majesty's Household

and for the County of London) and Mr. H. W. Wollaston (Barrister-at-Law of the Middle Temple).

The new edition incorporates the Coroners' Rules, 1953—detailed rules prescribing the practice and procedure to be followed by coroners and formulated for the first time in the 750-year-old history of the institution of coroners.

Entirely new sections (Chapters 6 and 7) deal with the meaning of violent, unnatural and sudden deaths, procedure at post-mortem examinations, medical evidence and fees for medical practitioners. These topics are of vital interest to all practising doctors.

The unique position of the coroner is well summarized by the authors in the Preface at pp. v-vi: 'Coroners are a good example of the English respect for tradition and their pragmatic approach to institutions. Coroners do not fit neatly into a logical system of judicial institutions. They have both judicial and executive functions though there is no proper system of appeal from their decisions: they straddle the domains of both the police and the medical profession though they are separate from each: and they are appointed not by the central government but by local authorities, though they are removable not by the local authorities (subject to certain exceptions) but by the Lord Chancellor.'

The vexed question of holding an inquiry into a post-operative death is set out with commonsense and clarity. 'It will be generally agreed that it is the duty of the medical profession to take the proper and necessary steps to fight disease and ill health and to save life. An operation is only one weapon in the armoury of medical science to be used when necessary; in other circumstances medical science may rely on drugs. Intervention by the medical profession in the form of an operation or other medical assistance may, therefore, be regarded as a natural concomitant or consequence to illness. The law holds that, if a man is deliberately shot and wounded, the fact that he dies under an operation properly performed to save his life, does not prevent a verdict of murder being returned. The chain of causation is not broken by the operation. The same approach should be adopted where an operation is performed on a person suffering from disease. If the operation is necessary and is properly performed, the operation should not necessarily be regarded as a *novus actus interveniens*. There will be occasions on which it could properly be so regarded: one would be if there were evidence of malpraxis on the part of the medical staff; another would be if the patient died from an abnormal reaction to an anaesthetic even though there was no suggestion of malpraxis. In the absence of such circumstances it would be legitimate to infer that the cause of death was the illness which necessitated the operation and that the operation was incidental' (pp. 90-91).

Unfortunately this reasonable view is not held universally and in some countries statutory requirements make *every* post-operative death the subject of a public inquest—a procedure which is often unfair to the surgeon and the anaesthetist involved and also very often prejudicial to the public interest in that it may create unnecessary and unwarranted fears in the public mind about submitting to surgical treatment.

The fairness of the procedure laid down for the holding of post-mortem examinations provides a model which certain other countries may well follow. It is often important for persons other than the medical practitioner performing the autopsy to be present at the examination. 'If it is known or suspected that any persons are likely to wish to be represented at the post-mortem examination, it is desirable that the post-mortem examination should be deferred until they are in a position to be represented thereat if such delay will not jeopardize the value of the examination' (p. 108). This is a sensible way to meet a situation which, in some other parts of the world, may require an order or the threat of an application to a court for an order to admit a medical practitioner to an autopsy in the capacity of an observer on behalf of interested persons.

The *Appendices* (pp. 255-554) set out the statutes, forms, etc. with which coroners may be concerned. They include a useful guide (with plans) for the construction of a mortuary with adequate regard not only to the requirements of the pathologist, but also to those of relatives and friends of deceased persons.

A very comprehensive index completes this outstanding and practical survey of the duties of the ancient office of coroners.

The Doctor as a Witness

The Doctor as a Witness. By John Evarts Tracy. 1st ed. 1957. (Pp. 217 + Index.) Philadelphia and London: W. B. Saunders Company.

The medical practitioner (both by training and experience) is particularly vulnerable when he appears in the un-

familiar surroundings of the law courts. He is usually ill-equipped to contend with the stratagems and devices to which he may be subjected. Nowhere in his medical curriculum is he instructed in the special problems that arise when he must give expert evidence in the courts and it is for this reason alone that Professor Tracy's volume entitled *The Doctor as a Witness* is most welcome.

Written simply and clearly illustrated with a wealth of pertinent examples, it should do much to fill the gaps in the medical practitioner's knowledge of how to set about the very special task of giving testimony.

The careful study of this volume will not only put the practitioner on guard against being trapped by trick questions in cross-examination, but will advance his ability to withstand cross-examination and, what is possibly even more important, to advise the legal practitioner with whom he is associated in the preparation for trial.

Professor Tracy quotes a telling description of what constitutes an expert witness, as stated by an eminent judge:

'No one has any title to respect as an expert, or has any right to give an opinion upon the stand, unless as his own opinion; and if he has not given the subject involved such careful and discriminating study as has resulted in the formation of a definite opinion, he has no business to give it. Such an opinion can only be safely formed or expressed by persons who have made the scientific questions involved matters of definite and intelligent study, and who have by such application made up their own minds. In doing so, it is their business to resort to such aids of reading and study as they have reason to believe contain the information they need. This will naturally include the literature of the subject. But if they have only taken trouble enough to find or suppose they find that certain authors say certain things, without further satisfying themselves how reliable such statements are, their own opinion must be of very moderate value, and whether correct or incorrect, cannot be fortified before a jury by statements of what those authors hold on the subject. The jury are only concerned to know what the witness thinks, and what capacity and judgment he shows to make his opinion worthy of respect' (pp. 53-54).

'Much of the contention and difficulty that arises between legal and medical practitioners, particularly when they are on opposite sides, stems from the totally different method of approach adopted towards a particular problem. This is well illustrated in the much publicized paternity suit involving Mr. Charles Chaplin. A medical practitioner would find the evidence of exclusion of paternity, when based on modern blood grouping tests, utterly convincing and completely unanswerable. Although this was the position in the case referred to, the jury was permitted to find on the evidence of the mother that Mr. Chaplin was the father of her child.

Professor Tracy's monograph is of particular importance to medical practitioners who become involved with forensic duties. It is, however, extremely useful also for the practising lawyer, who can gain from this book a very clear understanding of the doctor's difficulties.

Notes and News

Post-Graduate Course in Forensic Medicine

International Meeting on Legal Medicine at Lyon

Under the auspices of the Society of Legal Medicine and Criminology of France and the Association of Legal Medicine of Lyon, an International Meeting will be held (in the medium of the French language) at Lyon from 17-19 October 1957.

Those interested in attending should communicate with the Honorary Secretary, Prof. L. Roche, Laboratoire de Medecine Legale, Faculte de Medecine, 8 Avenue Rockefeller, Lyon, France.